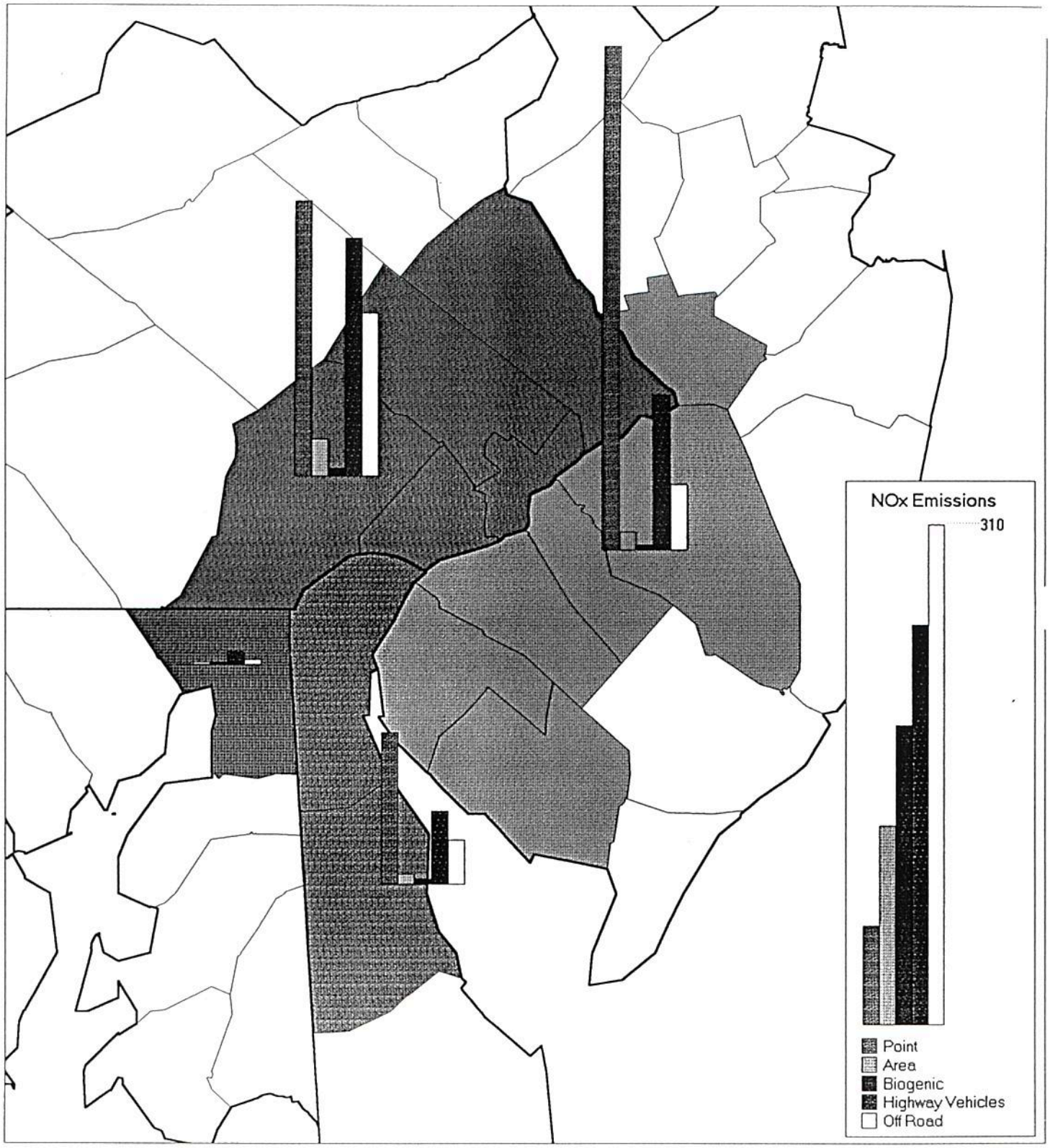
1990 VOC Emissions by State and Category
Philadelphia Nonattainment Area

1990 NOx Emissions by State and Category

Philadelphia Nonattainment Area



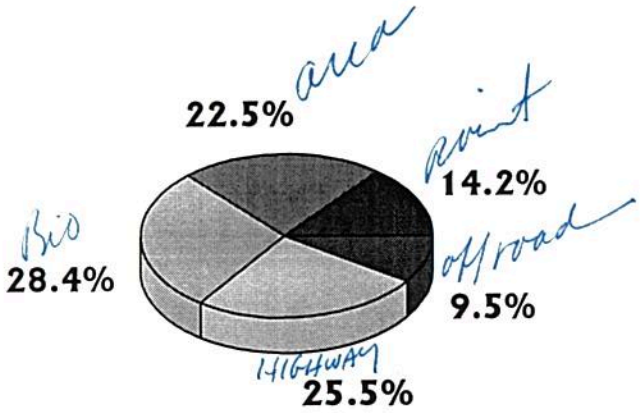
Top 20 VOC Emitters in 1990 in the Philadelphia Nonattainment Area

Top 20 NOx Emitters in 1990 in the Philadelphia Nonattainment Area

1990 VOC Emissions by County and Category
Pennsylvania Portion of Philadelphia Nonattainment Area

1990 NOx Emissions by County and Category
Pennsylvania Portion of Philadelphia Nonattainment Area

Philadelphia Nonattainment Area
1990 VOC Emissions by Source



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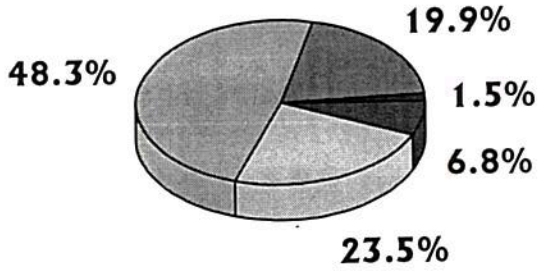

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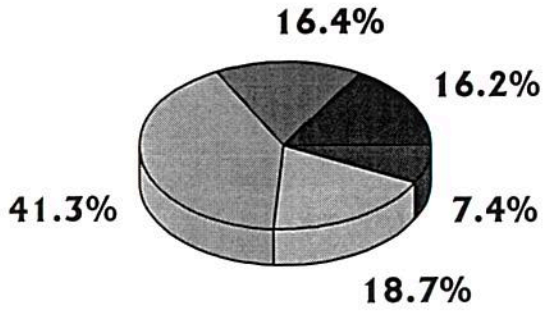

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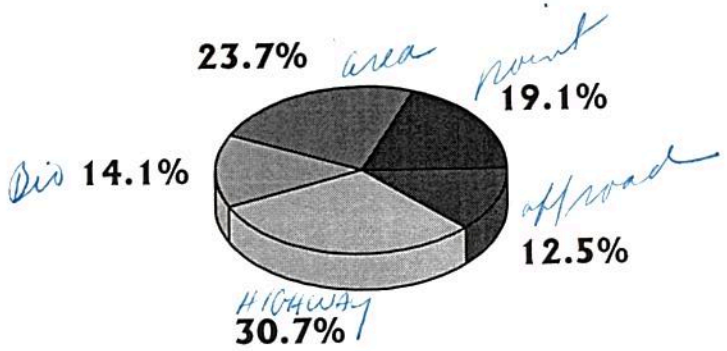
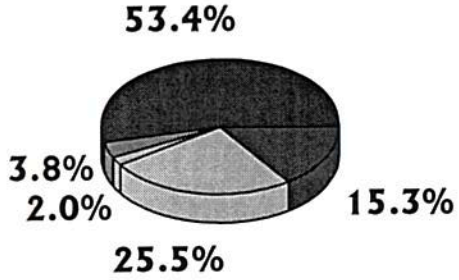


PA: 786 tpd

■ POINT SOURCES ■ AREA SOURCES ■ BIOGENIC ■ HWY VEHICLE ■ OFF ROAD

Philadelphia Nonattainment Area
1990 NOx Emissions by Source



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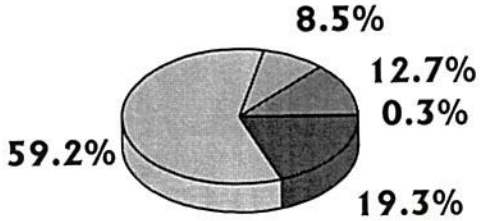

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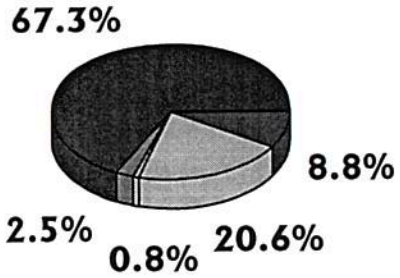

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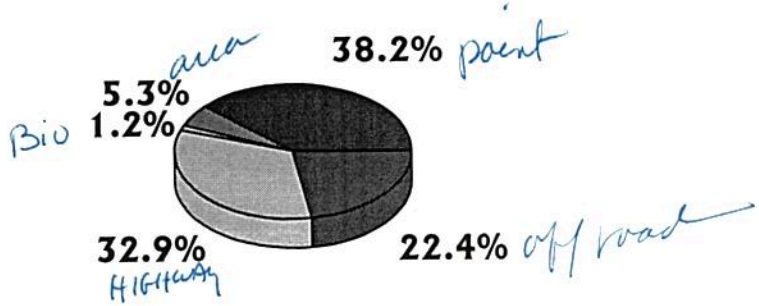

PA: 445 tpd

POINT SOURCES

AREA SOURCES

BIOGENIC

HWY VEHICLE

OFF ROAD

Measured Ozone in Philadelphia Area

- Presented to:
- Philadelphia Ozone Stakeholders Working Group, May 7, 1996
- Presented by:
- E.H. Pechan & Associates, Inc.

Measured Ozone in Philadelphia Area

- Presented to:
- Philadelphia Ozone Stakeholders Working Group, May 7, 1996
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Presentation Outline

- Ozone Monitoring Sites
- Definition of Terms
- Original Design Value Determination for Philadelphia
- Current Design Value Calculation
- Summary of Monitoring Data

Determination of Nonattainment Area Status

- Nonattainment area status determined by ozone design value
- 121-138 ppb = marginal nonattainment
- 138-160 ppb = moderate nonattainment
- 160-180 ppb = serious nonattainment
- 180-280 ppb = severe nonattainment
- 280+ ppb = extreme nonattainment

Daily Maximum Ozone Standard

- 0.12 parts per million (ppm) averaged over one hour
- Any measurement above 124 parts per billion (ppb) ozone is considered to be an ozone exceedance.

Definition of Design Value

- The daily maximum one-hour ozone concentration with the rank equal to the number of years of complete monitoring data plus one
- Also, the ozone concentration with the expected number of exceedances equal to one

Criteria for Valid Monitoring Data

- For values greater than the standard--all values considered valid regardless of number of hourly values available for that day

96

Ozone

Michael Lipsett, M.D.

INTRODUCTION

Ozone (O_3) is a naturally occurring colorless or light blue gas with a pungent "electrical" odor (1, 2). As a reactive oxidizing agent that is slightly soluble in water, ozone is a potent respiratory tract irritant. Since ozone is the principal oxidant found in photochemical smog, exposure occurs most commonly by breathing air in urban and suburban environments. Currently over half of the U.S. population lives in areas that have not met the federal ambient air quality standard for ozone (3). The continued failure to attain this clean air objective means that millions of people are intermittently exposed to ozone concentrations that would violate the occupational standard if such exposures were to occur in the workplace (See Table 96.1). Although ozone exposure may occur in a wide variety of occupational settings, published reports of accidental industrial intoxication are uncommon.

SITES, INDUSTRIES, AND BUSINESSES ASSOCIATED WITH EXPOSURE

Ozone occurs in the environmental and occupational settings listed in Table 96.2.

Mechanisms of Formation and Related Industrial Processes

Ozone in ambient air is formed by the action of ultraviolet solar radiation on nitrogen oxides and reactive hydrocarbons, both of which are emitted by motor vehicles and many industrial sources. Although the overall chemistry is complex, the basic reaction sequence involves the photodissociation of nitrogen dioxide (NO_2) into nitric oxide (NO) molecules and oxygen atoms. The latter react with oxygen (O_2) to form ozone. Because the reactions are driven by UV radiation, ozone formation tends to be greatest on warm, sunny days. The daily pattern of ambient ozone formation in heavily populated areas is typically characterized by a broad peak lasting from the late morning until the late afternoon or early evening (3). Ozone is also formed by the effect of lightning on oxygen in the atmosphere and at high altitudes by the action of ultraviolet light on oxygen.

Indoor ozone tends to reflect outdoor concentrations, but at substantially lower levels, owing to its ready destruction on indoor surfaces (7). The most common nonindustrial indoor sources are photocopying machines and electrostatic air cleaners (3). Electronic irradiation of air is used to manufacture ozone used commercially. Because of the high cost of shipping ozone, it is usually manufactured on-site (1). The most common occupational exposures to ozone have been reported to occur in electric arc welding, in industries using ozone as an oxidizing agent, and in aircraft cabins (8–10). However, during the 1980s

Table 96.1. Exposure Limits and Guidelines

Environmental	
National Ambient Air Quality Standard	0.12 ppm (1-hr avg)
Recommended Episode Criteria (Smog Alert Levels)	
Stage 1 (Alert)	0.20 ppm (1-hr avg)
Stage 2 (Warning)	0.40 ppm
Stage 3 (Emergency)	0.50 ppm
Emergency Exposure Limit (NAS)	1 ppm (1-hr avg)
Occupational	
Threshold Limit Value (ACGIH)	0.10 ppm (8-hr TWA)
Permissible exposure limit (OSHA)	0.10 ppm (8-hr TWA)
Short-term exposure limit (OSHA)	0.30 ppm (15-min avg)
Immediately dangerous to life and health (NIOSH)	10 ppm (30-min avg)

Sources: 40 Code of Federal Regulations 50 (1989); 29 Code of Federal Regulations 1910.1000 (1989); American Conference of Governmental Industrial Hygienists. Documentation of threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH: 1986:453; National Research Council, Committee on Toxicology. Emergency and continuous exposure limits for selected airborne contaminants. Vol 1. Department of Health and Human Services, Public Health Service. NIOSH pocket guide to chemical hazards. Washington, DC: US Government Printing Office, 1990:172.

Table 96.2. List of Sites, Uses, and Occurrence of Ozone

Environmental
Stratosphere (up to 10 ppm from UV effect on oxygen)
Troposphere (photochemical smog, electrical storms)
Occupational
Oxidizing agent in chemical manufacturing
Peroxide manufacturing
Disinfectant (drinking water, food in cold storage rooms, sewage treatment)
Deodorizing agent (air, sewer gas, feathers)
Industrial waste treatment
Bleaching agent (paper pulp, oils, textiles, waxes, flour, starch, sugar)
Aging of liquor and wood
Contamination of high altitude aircraft cabins
Mercury vapor lamps
Photocopy machines
Electric arc welding
High voltage electrical equipment
Linear accelerators
X-ray generators
Indoor ultraviolet sources
Electrostatic air cleaners

Sources: Sax NI, Lewis RJ. Hawley's condensed chemical dictionary. 11th ed. New York: Van Nostrand Reinhold Company, 1987; National Research Council, Committee on Indoor Pollutants. Indoor pollutants. Washington, DC: National Academy Press, 1981; Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR. Occupational diseases. A guide to their recognition. Washington, DC: U.S. Department of Health, Education, and Welfare, National Institute for Occupational Safety and Health, 1977:428–430.

aircraft ozone exposures were drastically reduced as airlines installed ozone converters in ventilation systems and began listing tropopause heights in flight plans (to alert pilots to modify flight paths, if necessary, to avoid cabin ozone contamination; personal communication, Dr. William Wells, United Air Lines). Given the magnitude of the ozone air pollution problem, persons in outdoor occupations, particularly those requiring physical exertion (see "Absorption," below), may also receive overtly toxic exposures.

CLINICAL TOXICOLOGY

Route of Exposure

Due to its high chemical reactivity, the half-life of ozone gas in liquid or solid media is negligible (11). Thus, ozone uptake is generally limited to anatomical sites of air-liquid interface (e.g., the mucous membranes of the respiratory tract and eye).

Absorption

Ozone is a strong irritant, and its relatively low solubility facilitates delivery to the lower respiratory tract, the principal target site. Still, ozone is absorbed throughout the respiratory tract. Although systemic absorption is limited by ozone's reactivity, a small fraction of inhaled ozone is absorbed into the blood, resulting in increased red blood cell fragility and alterations in blood chemistry (12).

Approximately 40–50% of inspired ozone is taken up in the nasopharynx, while about 90% of the ozone reaching the lower respiratory tract is removed (13, 14). Oral or oronasal (contrasted with exclusively nasal) breathing and a lower ventilation rate result in small, but statistically significant, increases in extrathoracic uptake of ozone in tidal-breathing human subjects. Similar modest increases in intrathoracic removal efficiency are associated directly with concentration and inversely with breathing rate (14). One model of ozone dosimetry predicts tissue penetration throughout the lung, with the greatest tissue dose occurring at the junction of conducting airways and gas exchange parenchyma, and a minute fraction absorbed into the blood (15, 16). These predictions are consistent with the distribution of lesions observed in several animal species. Recent work involving real-time measurements in the posterior pharynx of ozone-exposed volunteers suggests that reduction of tidal volume, a common functional response to ozone exposure, results in a significant decline in lower respiratory tract uptake of ozone, which is in reasonable quantitative agreement with the prediction of the above-noted model (17).

The magnitudes of symptomatic and functional responses to acute ozone exposure are roughly proportional to the effective dose delivered to the lung (i.e., concentration × duration of exposure × minute ventilation) (18, 19). There has been extensive documentation of enhanced responses to ozone associated with increasing concentration and ventilation (20). Only recently, however, has the importance of duration of exposure been quantified. Chamber studies involving exposures up to 6.6 hours in length with moderate exercise (ozone concentrations were $\leq$0.12 ppm) demonstrate a progressive increase in respiratory symptoms and a concomitant decline in pulmonary function indices (21, 22).

Metabolism

A potent oxidant, ozone is capable of reacting with many types of biological molecules and tissues, making it difficult to identify a characteristic critical biochemical effect. However, ozone's toxicity has been attributed primarily to oxidation of: (a) amino acids and sulfhydryl groups in enzymes and other proteins; and (b) polyunsaturated fatty acids to fatty acid peroxides, resulting in free radical formation (20). Cellular membranes contain both protein and lipid, and are thought to be the major site of action of ozone toxicity (23). Free radicals react with molecular oxygen to form organic peroxy free radicals, which in turn react with phospholipids in the cellular plasma membrane, resulting in denaturation of unsaturated fatty acid side chains and the creation of additional organic free radicals. Peroxidation of membrane structural lipids results in predictable toxic effects: increased permeability across the membrane, leakage of essential electrolytes and enzymes, inhibition of intracellular metabolic chains, and swelling and disintegration of mitochodria, lysozomes, and other organelles (24). Consistent with these observations, increased airway epithelial permeability due to ozone exposure has been reported in experimental animals and in human volunteers (25, 26). Severe damage results in cell lysis and necrosis, which has been observed in ozone-exposed experimental animals.

SIGNS, SYMPTOMS, AND SYNDROMES OF TOXIC EXPOSURE

Acute Toxicity

SYMPTOMS AND SIGNS OF ACUTE OZONE EXPOSURE

The most common respiratory symptoms caused by exposure to ambient levels of ozone are cough, substernal pain or soreness on deep inspiration, shortness of breath, chest tightness, dry throat, wheeze, and dyspnea (3). Nonrespiratory symptoms reported in controlled exposures of volunteers also include headache, nausea, and malaise. These effects are unlikely to occur in individuals at rest when ambient ozone concentrations are less than 0.30 ppm. However, as noted above, increasing the ventilation rate or duration of exposure can provoke symptoms at ozone concentrations as low as or even lower than the current federal ambient air quality standard (0.12 ppm, averaged over 1 hour) (21, 27). Earlier occupational case reports and a controlled study representative of occupational but not ambient exposures suggest a more severe spectrum of pulmonary and extrapulmonary effects, including (in addition to the above-noted symptoms) somnolence and extreme fatigue, dizziness, insomnia, decreased ability to concentrate, cyanosis, pulmonary edema, acrid taste and smell, and eye irritation (see below) (2, 28–31). Animals exposed to higher concentrations of ozone (3.2–12 ppm) for 4 hours die from pulmonary edema and hemorrhage (28, 32). In view of the dearth of published reports of severe respiratory outcomes in humans, however, exposures sufficient to induce them must be quite rare.

Substantial interindividual variability in sensitivity to ozone is common, but preexisting respiratory disease per se does not necessarily entail heightened toxic responses. For instance, in an investigation involving controlled 2-hour exposures to ozone and to filtered air, subjects with a history of allergic rhinitis

did not differ from normal subjects in ozone-related symptoms or pulmonary function changes, with the exception of a slightly greater increase in specific airway resistance (33). In contrast, several epidemiologic studies suggest that ozone concentrations found in urban air can provoke asthmatic episodes (34–36). Interestingly controlled exposure studies suggest that mild asthmatics do not appear to be markedly more sensitive to the effects of ozone than healthy individuals (37, 38, 38a). Nor does chronic obstructive pulmonary disease appear to enhance respiratory sensitivity to ozone (39, 40).

Ozone has also been well documented to significantly impair the ability to perform sustained exercise (41–43). Inspiratory discomfort is thought to be the principal reason for the diminution of exercise performance (44, 45).

Acute exposure to ozone also produces marked effects on pulmonary mechanics and bronchial reactivity. Established consequences of ozone exposure in chamber studies include decreases in inspiratory capacity, FVC, FEV_1, peak flow, and tidal volume, and increased specific airway resistance and frequency of respiration (20, 21, 32, 46, 46a). Some, but not all, of these responses can be blocked by pretreatment with atropine, and thus are thought to be mediated by the parasympathetic nervous systems possibly through reflex inhibition of inspiratory muscle contraction (46, 46a, 47). Increased airway reactivity after ozone exposure is associated with significant increases of arachidonic acid metabolites and neutrophils in the airways, indicating the potential importance of inflammation as both a consequence and a mediator of ozone toxicity in humans and experimental animals (48–50). There is considerable interindividual variability in functional responsiveness to ozone, with 5–25% of study populations demonstrating markedly greater effects than other subjects (20). That the functional changes are highly reproducible over periods from 3 weeks to 14 months suggests the existence of an intrinsic responsiveness to ozone (51, 52).

Several field studies of children suggest that exposure to ozone concentrations at or below the federal ambient air quality standard is associated with transient decrements in lung function (53, 54). In one study of an air pollution episode lasting several days, during which maximum daily ozone concentrations ranged between 0.12 and 0.185 ppm, peak flow decrements in some children lasted up to a week after termination of the episode (55). In a few controlled exposure studies, children appear to experience declines in pulmonary function comparable in magnitude to those observed in adults, but they do not report symptoms to the same extent (56–58). Although this apparent difference in symptom reporting between children and adults may represent real differences in somatic perception, it may also be the result of the relatively low mean ozone concentrations to which the children were exposed.

In some individuals, acute symptomatic and functional responses to ozone become attenuated with repeated daily exposures. In controlled chamber studies, maximum responses are observed on the second day of exposure, but on subsequent days there may be little or no ozone-related effect (59). In a laboratory setting, "adaptation" to ozone toxicity typically persists for up to 1 week following cessation of exposure but may last up to about 3 weeks (60–62). Repeated real-world exposures appear to induce longer periods of attenuated responses (62a).

Eye irritation that occurs during smog episodes is due mainly to other photochemical oxidants, such as peroxyacetylnitrate, not to ozone (20, 63–64). However, in industrial settings, eye and nasal irritation may occur (65). Concentrations greater than 2 ppm have been reported to be irritating to normal human eyes within minutes (66).

Numerous studies of mice exposed even briefly (2–3 hours) to ozone concentrations at or below the current federal ambient air quality standard (0.12 ppm) have shown significantly decreased resistance to bacterial but not viral respiratory infections (67–72). A limited number of epidemiologic and clinical studies have, in general, failed to detect an effect of ozone or oxidant air pollution on respiratory infections in humans, although this issue has not been adequately investigated (73–76).

PATHOLOGY

Ozone may damage tissues throughout the respiratory tract, depending on the pattern of breathing and the exposure concentration and duration. At high concentrations, ozone may cause desquamation of the airways and pulmonary edema (77). At sublethal concentrations (up to 1.0 ppm), airway epithelial cells are also damaged, but the principal site of injury is the central portion of the pulmonary acinus. Type I alveolar and ciliated bronchiolar cells appear to be particularly susceptible to ozone toxicity, with damage evident as early as four hours of exposure (78). Inflammatory responses at the junction of the conducting airways and the gas exchange zone have been reported consistently in studies of rodents, dogs, and nonhuman primates (20). Continued exposure over several days results in replacement of type I by type II cells as well as hypertrophy and hyperplasia of nonciliated cuboidal cells in the bronchiolar epithelium (79, 80). When animals are allowed to recover in clean air from acute and subacute exposures, these lesions all appear to be reversible (79, 80).

Although microscopic examination of airway damage from acute ozone exposure has not been performed in humans, several investigators have measured markers of inflammation in ozone-exposed volunteers. Bronchoalveolar lavage fluid from these subjects showed large increases in polymorphonuclear cells (up to 8.2-fold over control levels), other inflammatory mediators, and protein concentrations consistent with a transudation of serum (48, 81). The latter finding suggests increased pulmonary vascular permeability, one of the hallmarks of inflammation, and tends to corroborate earlier work demonstrating increased permeability of the respiratory epithelium, as measured by ^{99}Tc-labeled DTPA clearance (25). Although these data are somewhat limited, they indicate that the inflammatory effects of ozone repeatedly demonstrated in animals also occur in the human lung.

Chronic Toxicity

One of the principal uncertainties about ozone toxicity is the relationship between repeated exposures and chronic respiratory disease. Exposure of guinea pigs and rats to a relatively high ozone concentration (approximately 1.0 ppm) for 268 days caused a chronic bronchiolitis, with bronchiolar fibrosis, pneumonitis, "mild to moderate" emphysema, and occasional epithelial lesions in the trachea and major bronchi (82). Exposure of rats to substantially lower concentrations (between 0.12 and 0.25 ppm) resulted in less severe but still significant changes in the terminal bronchioles and alveolar septa, as well as a distribution of inflammation similar to that observed in acute exposures (83, 84). Chronic exposures of monkeys showed

changes in nasal epithelial secretory product, respiratory bronchiolitis (with inflammatory thickening of the bronchiolar wall and hypertrophy and hyperplasia of nonciliated cuboidal cells in the bronchiolar epithelium), and other changes, including the development of hyperplastic nodules that persisted after the cessation of exposure (85–87). It is noteworthy that in a 3-month study of nonhuman primates, the degree of inflammation after 90 days was less than that observed after 7 (85). This may be a consequence of the greater resistance of the altered epithelial cell population to environmental insults.

These animal experiments demonstrate that chronic exposure to ozone concentrations found in typical urban air results in centriacinar inflammation and small airway structural changes. Other lines of evidence support the notion that repeated ozone exposure may result in chronic lung disease, including the observations that ozone inactivates human alpha-1 antiproteinase inhibitor and appears to cause the synthesis and deposition of abnormal collagen in rat lung (88, 89). Recent epidemiologic studies suggest the existence of significant associations of photochemical oxidant exposure with an accelerated decline in lung function and with symptoms of chronic respiratory disease in nonsmokers (90, 91). Problems in longterm measurement of ozone or oxidant exposure and the high covariation between ozone and particulate air pollution, however, limit the interpretation of these investigations.

Genetic Toxicity and Carcinogenicity

Ozone is genotoxic in a variety of assay systems, but results of different experiments are inconsistent (20). Effects reported include bacterial mutations, plasmid DNA strand breakage, sister chromatid exchange, and chromatid and chromosome breaks in lymphocytes (92). For example, a threefold increase in chromatid-type aberrations persisted for up to 6 weeks in subjects exposed to 0.5 ppm ozone for 6–10 hours (93). In contrast, no significant changes in chromosome or chromatid breaks were observed in lymphocytes of subjects exposed to 0.4 ppm ozone for 4 hours (94). More recently, cultured human epidermal cells exposed to 5 ppm ozone for 10 minutes showed no indication of any DNA strand breakage (95). Although ozone's ability to cause free radical formation gives grounds for suspicion that it may be genotoxic in humans, this issue has not been extensively explored (95a).

Short (5-minute) exposure to 5 ppm ozone induces neoplastic transformation in hamster embryo cells and mouse fibroblasts (96). Although some studies suggest that chronic ozone exposure may cause the development of murine pulmonary adenomas and other hyperplastic nodules in the lungs of nonhuman primates, this compound has not been adequately tested for carcinogenicity (86, 97). However, because exposure to ozone is so common and because there is some experimental documentation of oncogenicity, the U.S. National Toxicology Program has selected ozone to be tested in a 2-year carcinogenesis bioassay, which is ongoing at the time of this writing.

Management of Toxicity

Avoidance of exposure is obviously the best management strategy. In the occupational setting this means providing adequate engineering controls (e.g., entirely enclosed processes or local exhaust ventilation), thorough worker education about appropriate work practices (use of personal protective equipment, such as an ozone-decomposing respirator, when adequate ventilation is impractical) and recognition of ozone-related symptoms, and strict adherence to health and safety rules. In the context of environmental exposures, individuals should be advised to avoid doing aerobic exercise during peak ozone hours (typically late morning until early evening in many urban areas) and to pay attention to the health advisories accompanying the declaration of a smog alert. However, it should be borne in mind that signs and symptoms of ozone toxicity have been repeatedly demonstrated to occur in exercising adults at ozone concentrations lower than the current recommended stage 1 smog alert level (0.20 ppm) (21, 22, 32).

Diagnosis of ozone-related toxicity is based on a history of exposure and recognition of symptoms compatible with exposure. Because ozone symptomatology may mimic several cardiorespiratory illnesses, the differential diagnosis includes influenza, the common cold, sinusitus, asthma, bronchopneumonia, pulmonary embolism, and myocardial infarction (30). Asthmatic episodes triggered by ozone should be treated according to standard protocols. Although ozone is theoretically capable of causing pulmonary edema in humans, the scarcity of published reports indicates that it is historically rare. Severe industrial overexposure should be managed like other acute inhalational injury, with supportive treatment. Except in these unusual instances, ozone-related symptoms are self-limited after termination of exposure, with recovery in milder cases generally occurring within hours. Symptomatic treatment would include analgesics for headache and chest pain and cough suppressants if indicated. Some reports of industrial ozone toxicity indicate a more prolonged convalescence, with resolution of symptoms occurring over 1–2 weeks (30).

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AMERICAN LUNG ASSOCIATION®

Out Of Breath:

Populations-at-Risk to Alternative Ozone Levels

American Lung Association • 1726 M St., NW, Suite 902 • Washington, D.C. 20036-4502
November 1995

SUMMARY OF FINDINGS

This report presents estimates of the total number of people in the United States, as well as those who are at greatest risk from exposure to ozone air pollution (children, the elderly, people with asthma and chronic obstructive pulmonary disease) and live in areas that violate either the current federal ozone standard (0.12 parts per million) or two alternative standard levels. The two alternative ozone levels selected for this report are based on the top and bottom of the range of alternative eight-hour average ozone standard levels currently under consideration by EPA as a revised national ozone standard.

One alternative under EPA consideration is a 0.07 ppm, one-exceedance level, which the American Lung Association supports as providing the most public health protection with the margin of safety required by the Clean Air Act. This report also estimates the number of people that would be covered by a 0.09 ppm, five-exceedance standard, the least protective alternative under EPA consideration. Data on the numbers of at-risk people covered by the current ozone standard are included for sake of comparison.

Table 1 of the report summarizes the national statistics for at-risk populations for the current 0.12 ppm ozone standard and the two alternative standard scenarios. Table 2 provides total population statistics by state for the three ozone standard levels. Tables 3, 4 and 5 provide state totals of the at-risk population categories. Tables 6, 7 and 8 provide county level estimates of at-risk and total populations.

Major findings of the report are:

An estimated 161 million people, representing 63 percent of the U.S. population, live in areas that exceed the 0.07 ppm, one-exceedance ozone standard alternative under consideration by EPA. These people are potentially exposed to **unhealthful** ozone levels.

An estimated 33 million children, 20 million elderly, 8 million people with asthma and 9 million people with chronic obstructive lung disease live in areas that exceed the 0.07 ppm ozone level. These people are potentially exposed to **unhealthful** ozone levels.

The number of at-risk people protected by the most lax ozone standard (0.09 ppm, five exceedances) under consideration by EPA is almost 75 percent less than the more protective standard (0.07 ppm, one exceedance) recommended by American Lung Association.

Even though the level of the 0.09 ppm standard alternative is lower than the current 0.12 ppm ozone standard, allowing multiple exceedances of this level results in a 30 percent reduction in the number of people protected even when compared to the inadequate current ozone standard.

TABLE 6: ESTIMATED POPULATIONS-AT-RISK LIVING IN COUNTIES WITH ONE OR MORE ANNUAL EXCEEDANCES OF A 0.07 ppm EIGHT HOUR AVERAGE OZONE LEVEL

		POPULATIONS-AT-RISK						
		CHRONIC DISEASE			AGE (YEARS)			
COUNTY	OZONE LEVEL (1)	COPD (2)	ADULT ASTHMA	PEDIATRIC ASTHMA	<5	5-13	65+	TOTAL POPULATI
MONTGOMERY CO.	0.10	38,562	22,520	11,280	48,995	82,198	95,109	689,996
NORTHAMPTON CO.	0.10	14,106	8,238	4,126	17,922	30,067	34,790	252,393
PERRY CO.	0.10	2,370	1,384	693	3,011	5,052	5,845	42,406
PHILADELPHIA CO.	0.11	86,769	50,673	25,382	110,243	184,956	214,006	1,552,572
WASHINGTON CO.	0.10	11,516	6,725	3,369	14,631	24,547	28,402	206,054
WESTMORELAND CO.	0.09	20,919	12,216	6,119	26,578	44,590	51,593	374,300
YORK CO.	0.10	19,557	11,421	5,721	24,848	41,687	48,234	349,932
TOTALS:		495,755	289,519	145,021	629,874	1,056,745	1,222,717	8,870,594
RHODE ISLAND								
KENT CO.	0.12	9,169	5,477	2,393	11,510	18,662	24,860	162,493
PROVIDENCE CO.	0.09	33,326	19,908	8,698	41,833	67,827	90,355	590,591
TOTALS:		42,495	25,385	11,091	53,342	86,488	115,215	753,084
SOUTH CAROLINA								
ABBEVILLE CO.	0.08	1,319	784	400	1,824	3,120	2,783	24,072
AIKEN CO.	0.08	7,043	4,185	2,138	9,744	16,665	14,861	128,566
ANDERSON CO.	0.09	8,123	4,827	2,466	11,238	19,219	17,140	148,275
BARNWELL CO.	0.09	1,155	687	351	1,598	2,734	2,438	21,089
BERKELEY CO.	0.08	7,460	4,433	2,265	10,321	17,652	15,742	136,184
CHARLESTON CO.	0.08	16,685	9,915	5,065	23,084	39,479	35,207	304,578
CHEROKEE CO.	0.09	2,498	1,484	758	3,456	5,911	5,271	45,602
CHESTER CO.	0.09	1,789	1,063	543	2,475	4,233	3,775	32,659
DARLINGTON CO.	0.08	3,486	2,072	1,058	4,823	8,249	7,357	63,642
EDGEFIELD CO.	0.09	1,022	607	310	1,414	2,419	2,157	18,660
OCONEE CO.	0.09	3,231	1,920	981	4,470	7,645	6,817	58,978
PICKENS CO.	0.09	5,404	3,211	1,641	7,477	12,787	11,403	98,652
RICHLAND CO.	0.10	16,106	9,571	4,889	22,283	38,109	33,985	294,004
SPARTANBURG CO.	0.09	12,757	7,581	3,873	17,650	30,185	26,919	232,875
UNION CO.	0.08	1,675	995	508	2,318	3,964	3,535	30,578
WILLIAMSBURG CO.	0.09	2,030	1,206	616	2,808	4,803	4,283	37,052
YORK CO.	0.11	7,517	4,467	2,282	10,399	17,785	15,861	137,211
TOTALS:		99,300	59,007	30,143	137,384	234,958	209,533	1,812,677
TENNESSEE								
ANDERSON CO.	0.09	3,919	2,336	1,110	4,968	8,687	8,986	70,525
BLOUNT CO.	0.09	5,023	2,995	1,422	16,368	11,136	11,519	90,403
DAVIDSON CO.	0.08	28,773	17,153	8,147	36,474	63,781	65,976	517,798
FAYETTE CO.	0.08	1,444	861	409	1,831	3,202	3,312	25,995
HAMILTON CO.	0.09	16,039	9,562	4,542	20,332	35,554	36,777	288,637
HAYWOOD CO.	0.10	1,082	645	306	1,372	2,399	2,481	19,474
JEFFERSON CO.	0.10	1,932	1,152	547	2,449	4,283	4,430	34,770
KNOX CO.	0.10	19,314	11,514	5,469	24,484	42,814	44,288	347,583
MADISON CO.	0.08	4,458	2,658	1,262	5,651	9,883	10,223	80,230
MAURY CO.	0.08	3,320	1,979	940	4,208	7,359	7,612	59,740
RUTHERFORD CO.	0.08	7,153	4,265	2,025	9,068	15,857	16,403	128,731
SEVIER CO.	0.09	3,038	1,811	860	3,851	6,734	6,966	54,670
SHELBY CO.	0.10	46,946	27,987	13,293	59,512	104,066	107,648	844,847
SULLIVAN CO.	0.09	8,150	4,859	2,308	10,332	18,067	18,689	146,676
SUMNER CO.	0.11	5,998	3,576	1,698	7,603	13,295	13,753	107,937
WILLIAMSON CO.	0.09	4,925	2,936	1,395	6,244	10,918	11,294	88,640
WILSON CO.	0.09	3,954	2,357	1,120	5,013	8,765	9,067	71,160
TOTALS:		165,469	98,647	46,854	209,761	366,800	379,424	2,977,816
TEXAS								
BEXAR CO.	0.09	66,365	38,828	22,480	104,717	177,685	125,282	1,233,096
BRAZORIA CO.	0.11	10,971	6,419	3,716	17,312	29,375	20,712	203,857
COLLIN CO.	0.10	15,655	9,159	5,303	24,702	41,914	29,552	290,873
DALLAS CO.	0.10	102,978	60,249	34,883	162,489	275,714	194,399	1,913,395
DENTON CO.	0.11	15,863	9,281	5,373	25,031	42,473	29,946	294,750
ELLIS CO.	0.09	4,741	2,774	1,606	7,481	12,693	8,950	88,087
EL PASO CO.	0.09	33,824	19,789	11,457	53,371	90,561	63,852	628,472
GALVESTON CO.	0.12	12,275	7,182	4,158	19,369	32,866	23,173	228,084
GREGG CO.	0.09	5,810	3,399	1,968	9,167	15,555	10,967	107,945
HARDIN CO.	0.09	2,340	1,369	793	3,693	6,266	4,418	43,487
HARRIS CO.	0.13	159,938	93,575	54,177	252,368	428,221	301,928	2,971,755
JEFFERSON CO.	0.11	13,092	7,660	4,435	20,658	35,053	24,715	243,257
KAUFMAN CO.	0.09	2,929	1,714	992	4,622	7,842	5,529	54,424
NUECES CO.	0.09	16,190	9,472	5,484	25,546	43,347	30,563	300,815
ORANGE CO.	0.11	4,471	2,616	1,515	7,055	11,972	8,441	83,080

TABLE 6: ESTIMATED POPULATIONS-AT-RISK LIVING IN COUNTIES WITH ONE OR MORE ANNUAL EXCEEDANCES OF A 0.07 ppm EIGHT HOUR AVERAGE OZONE LEVEL

COUNTY	OZONE LEVEL (1)	POPULATIONS-AT-RISK						
		CHRONIC DISEASE			AGE (YEARS)			TOTAL POPULATION
		COPD (2)	ADULT ASTHMA	PEDIATRIC ASTHMA	<5	5-13	65+	
NORTH CAROLINA								
MECKLENBURG CO.	0.10	29,723	17,910	8,339	38,934	63,949	66,513	537,735
NEW HANOVER CO.	0.09	7,064	4,257	1,982	9,254	15,199	15,809	127,808
PERSON CO.	0.08	1,711	1,031	480	2,241	3,681	3,828	30,952
PITT CO.	0.09	6,214	3,744	1,744	8,140	13,370	13,906	112,426
ROCKINGHAM CO.	0.09	4,805	2,895	1,348	6,294	10,338	10,752	86,927
WAKE CO.	0.10	25,268	15,225	7,089	33,099	54,364	56,544	457,138
YANCEY CO.	0.09	866	522	243	1,135	1,864	1,939	15,673
TOTALS:		163,329	98,417	45,826	213,949	351,409	365,498	2,954,921
OHIO								
ALLEN CO.	0.09	6,140	3,606	1,792	7,984	14,235	14,563	110,179
ASHTABULA CO.	0.10	5,625	3,303	1,642	7,313	13,040	13,340	100,924
BUTLER CO.	0.10	17,000	9,983	4,962	22,105	39,412	40,319	305,041
CLARK CO.	0.10	8,242	4,840	2,406	10,717	19,108	19,548	147,891
CLERMONT CO.	0.09	8,814	5,176	2,573	11,461	20,435	20,905	158,161
CLINTON CO.	0.11	2,044	1,201	597	2,658	4,740	4,849	36,685
CUYAHOGA CO.	0.12	78,647	46,182	22,956	102,263	182,332	186,528	1,411,209
FRANKLIN CO.	0.10	55,290	32,467	16,138	71,892	128,182	131,132	992,095
HAMILTON CO.	0.11	48,598	28,537	14,185	63,191	112,668	115,261	872,026
JEFFERSON CO.	0.10	4,437	2,606	1,295	5,770	10,288	10,524	79,623
KNOX CO.	0.10	2,702	1,586	789	3,513	6,264	6,408	48,478
LAKE CO.	0.10	12,285	7,214	3,586	15,974	28,481	29,136	220,436
LAWRENCE CO.	0.11	3,517	2,065	1,027	4,573	8,153	8,341	63,105
LICKING CO.	0.10	7,355	4,319	2,147	9,564	17,052	17,444	131,975
LOGAN CO.	0.10	2,435	1,430	711	3,167	5,646	5,776	43,701
LORAIN CO.	0.09	15,419	9,054	4,501	20,049	35,748	36,570	276,679
LUCAS CO.	0.10	25,720	15,103	7,507	33,443	59,628	61,000	461,508
MADISON CO.	0.09	2,171	1,275	634	2,823	5,033	5,149	38,952
MAHONING CO.	0.10	14,802	8,692	4,321	19,247	34,317	35,107	265,607
MEDINA CO.	0.10	7,162	4,206	2,090	9,313	16,604	16,986	128,513
MIAMI CO.	0.09	5,288	3,105	1,544	6,876	12,261	12,543	94,894
MONTGOMERY CO.	0.10	32,248	18,936	9,413	41,931	74,762	76,483	578,642
PORTAGE CO.	0.10	8,148	4,785	2,378	10,595	18,891	19,325	146,209
PREBLE CO.	0.09	2,280	1,339	665	2,964	5,285	5,407	40,904
STARK CO.	0.10	20,739	12,178	6,053	26,966	48,080	49,186	372,125
SUMMIT CO.	0.11	29,158	17,122	8,511	37,913	67,598	69,153	523,191
TRUMBULL CO.	0.10	12,807	7,520	3,738	16,653	29,691	30,375	229,805
TUSCARAWAS CO.	0.08	4,766	2,799	1,391	6,197	11,049	11,303	85,518
UNION CO.	0.10	1,878	1,103	548	2,441	4,353	4,453	33,690
WARREN CO.	0.10	6,677	3,921	1,949	8,682	15,481	15,837	119,816
WASHINGTON CO.	0.11	3,498	2,054	1,021	4,548	8,110	8,296	62,766
TOTALS:		455,894	267,706	133,067	592,787	1,056,925	1,081,249	8,180,348
OKLAHOMA								
CLEVELAND CO.	0.08	10,129	5,841	3,080	13,188	24,900	24,604	181,388
McCLAIN CO.	0.08	1,310	756	399	1,706	3,221	3,183	23,467
OKLAHOMA CO.	0.09	34,214	19,730	10,405	44,549	84,110	83,111	612,713
TULSA CO.	0.10	29,029	16,739	8,828	37,797	71,362	70,514	519,847
TOTALS:		74,683	43,065	22,713	97,241	183,594	181,412	1,337,415
OREGON								
CLACKAMAS CO.	0.10	16,593	9,690	4,854	21,082	39,067	40,925	296,905
JACKSON CO.	0.08	8,612	5,029	2,519	10,941	20,275	21,240	154,090
LANE CO.	0.08	16,256	9,493	4,755	20,654	38,272	40,093	290,866
TOTALS:		41,461	24,213	12,128	52,677	97,614	102,258	741,861
PENNSYLVANIA								
ALLEGHENY CO.	0.11	74,576	43,552	21,815	94,751	158,965	183,932	1,334,396
BEAVER CO.	0.10	10,544	6,157	3,084	13,396	22,475	26,005	188,659
BERKS CO.	0.11	19,177	11,199	5,610	24,365	40,877	47,298	343,135
BLAIR CO.	0.09	7,339	4,286	2,147	9,325	15,644	18,101	131,319
BUCKS CO.	0.11	31,089	18,156	9,094	39,500	66,269	76,677	556,279
CAMBRIA CO.	0.10	9,059	5,290	2,650	11,510	19,310	22,343	162,096
DAUPHIN CO.	0.10	13,526	7,899	3,957	17,185	28,832	33,361	242,025
DELAWARE CO.	0.11	30,711	17,935	8,984	39,019	65,462	75,744	549,506
ERIE CO.	0.09	15,627	9,126	4,571	19,855	33,310	38,542	279,615
LACKAWANNA CO.	0.11	12,155	7,098	3,556	15,443	25,909	29,978	217,484
LANCASTER CO.	0.10	24,279	14,179	7,102	30,847	51,753	59,881	434,425
LAWRENCE CO.	0.09	5,393	3,149	1,577	6,851	11,495	13,300	96,489
LEHIGH CO.	0.10	16,544	9,662	4,840	21,020	35,265	40,804	296,027
LUZERNE CO.	0.10	18,383	10,736	5,377	23,356	39,185	45,339	328,927
LYCOMING CO.	0.08	6,733	3,932	1,969	8,554	14,351	16,605	120,468
MERCER CO.	0.10	6,823	3,985	1,998	8,669	14,545	16,829	122,091

facts about

Smog and California Crops

California has the world's richest and most productive farmland. California farmers produce an average of $17 billion in crops each year for their efforts. The state's agriculture provides about one-half of the nation's fruits and vegetables and one job out of every six to California's economy. In addition, related industries such as packing, canning, textiles and machinery make the total value of California agriculture more than $70 billion a year.

But California's number one industry may be losing more than $300 million each year to air pollution. Smog damage is a major reason why crops such as spinach, celery, lettuce, tomatoes, string beans and cucumbers, as well as many ornamental plants, are no longer grown ...mmercially around metropolitan Los Angeles.

... addition to crops, smog damages forests, range and ...sturelands that produce another $700 million in revenue for California each year. These natural ecosystems account for approximately 85 percent of California's land area and provide Californians with recreation and watershed land as well as supporting timber and livestock industries.

HISTORY

During the 1940's Southern California researchers were puzzled by what was called the "X" disease, that damaged trees, but could not be traced to its sources. By the early 1950's, however, a clear link had been established between Los Angeles smog and the mysterious plant disease. Studies confirmed that pollutants emitted from Los Angeles area factories and freeways were blown to farming areas downwind.

By the mid 1950's, smog damage to crops was being reported near Bakersfield and Fresno and ten years later, crop damage from smog was apparent in the state's most important agricultural regions.

California's 33 million acres of forestland have not escaped air pollution damage. Extensive injury to trees was discovered in the San Bernardino Mountains during the 1960's and tree injury in other Southern California national parks and in the Sierra Nevada was first reported during the 1970's.

HOW WEATHER, GEOGRAPHY AND AIR POLLUTION AFFECT VEGETATION

California's unique air pollution causes severe damage to vegetation. The mountains surrounding our valleys form basins that trap and hold air pollution. As pollution spreads throughout these valleys, temperature inversions, layers of air which trap air pollution beneath them, put a lid on those valleys preventing the air pollution from escaping. That air pollution can then "bake" under the state's sunny skies and be converted into ozone.

Nearly all of the state's major crop producing areas are located in those valleys or basins. Furthermore, that air pollution is able to climb the valley walls and carry air pollution to the mountain regions, where it can damage trees and grasses above the valleys.

OZONE STANDARD

California reduced its ozone standard in 1987 from .10ppm to .09ppm (25 percent more stringent than the federal health standard of .12ppm) to better protect the state's crops and natural vegetation from the effects of continued ozone exposure. Standards that are expected to protect public health, are also expected to lower vegetation damage.

WHICH POLLUTANTS DAMAGE PLANTS AND HOW

Air pollution interferes with photosynthesis, the process by which plants use sunlight to convert water and carbon dioxide to food and plant fiber. It can cause leaves to yellow and to develop dead areas, reducing photosynthesis. Air pollution reduces cash crop yield, carbohydrate content and visual appeal. Smog can also make vegetation more vulnerable to injury from diseases or pests.

AIR POLLUTION AFFECTS PLANTS BY:

- **Injuring leaves, stems and roots,**
- **reducing yield, cutting fruit size and weight,**
- **cutting market value by spotting leaves and fruit,**
- **causing plant death.**

OZONE

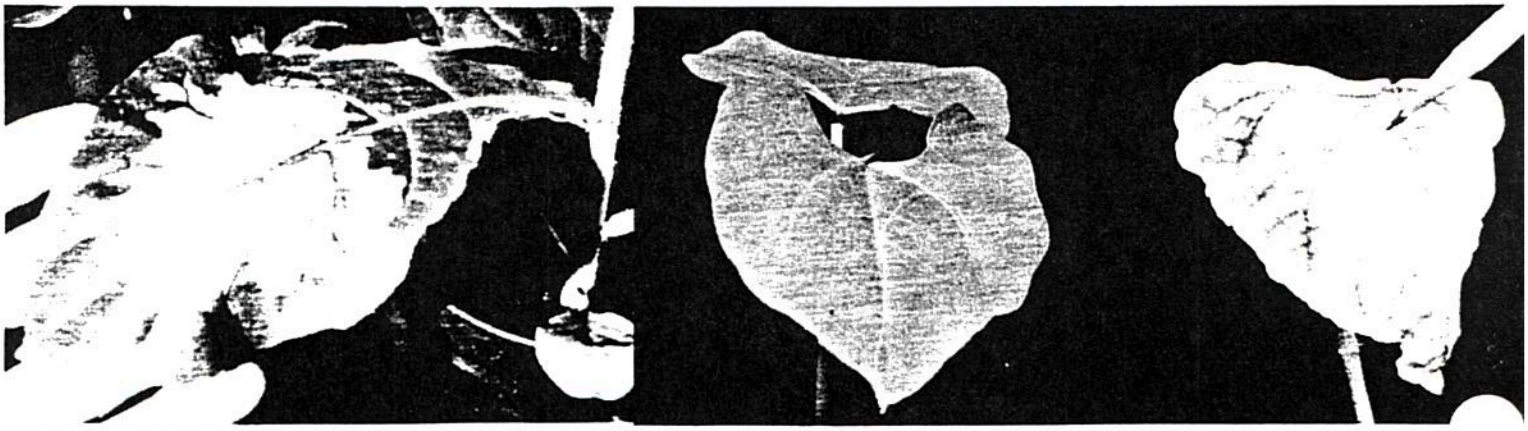
Ozone is created when hydrocarbons and nitrogen oxide emissions from motor vehicles and industr polluters react chemically with sunlight. It is also California's greatest air pollution problem and its most serious threat to vegetation. Ozone attacks leaves, causing them to yellow, develop dead spots and drop early. Low level ozone exposure over long time periods can reduce a plant's growth and fruit yield and increase its susceptibility to disease and insect attack.

SULFUR DIOXIDE

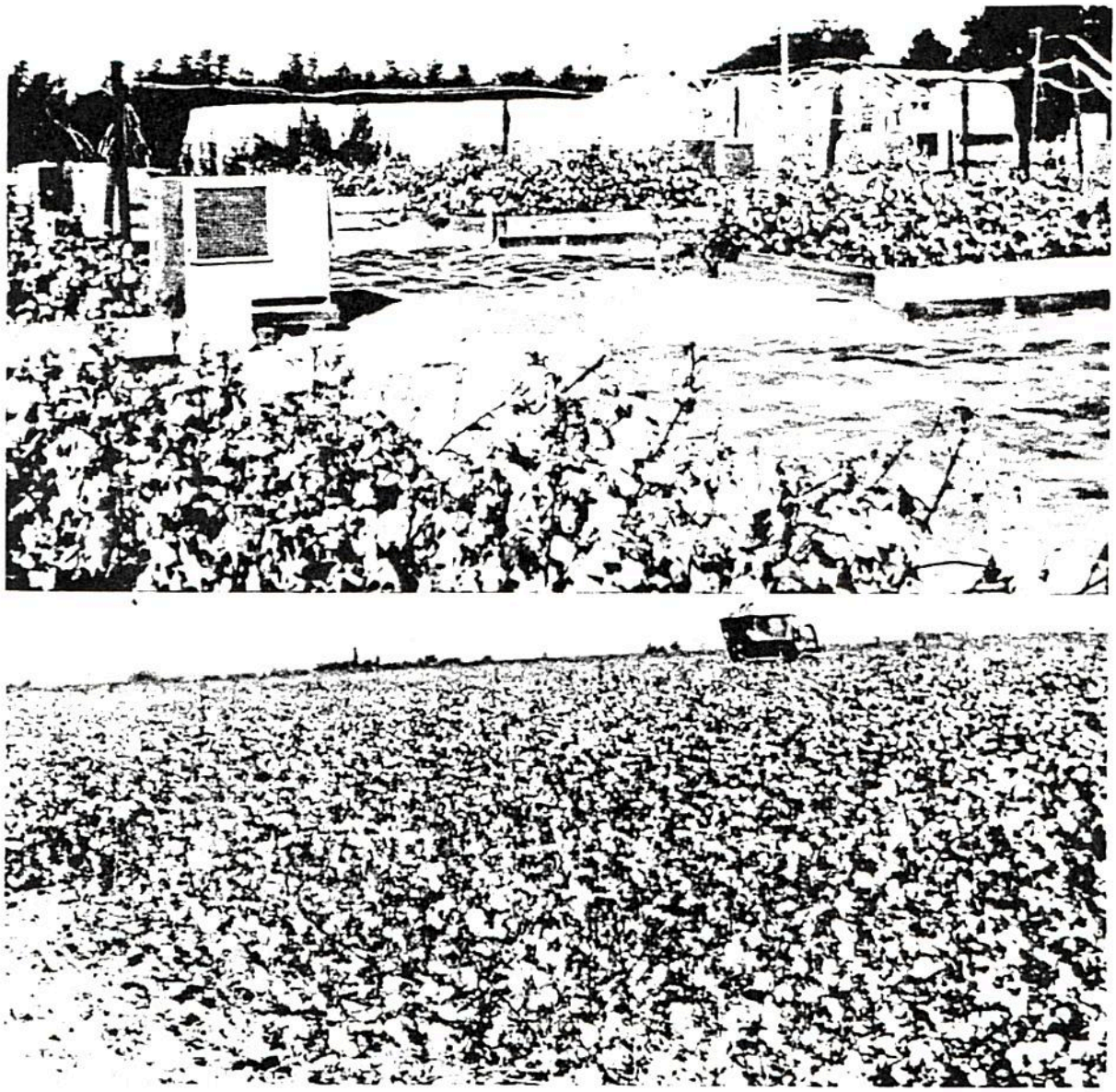
ARB research shows that sulfur dioxide (SO_2), which is mainly released from industrial sources such as factories and steam generators while burning coal or oil, may also damage plants. Short-term, high concentration sulfur dioxide exposure to vegetation can reduce root and stem weight, as well as cut protein and carbohydrate content and ultimately result in plant death.

OTHER POLLUTANTS

Other less common pollutants also affect vegetation. Flourides, ammonia and ethylene, by-products of industrial processes, as well as boron and hydrogen sulfide, emitted from geothermal operations, can injure leaves and reduce plant growth.



Discolored leaves are a result of air pollution that can damage crops and reduce yield.



Research on cotton yields has been conducted both under laboratory and field conditions.

MAJOR CALIFORNIA CROPS AFFECTED BY AIR POLLUTION

The effects of air pollution on several California crops and plants have been documented during the last decade through experiments in which crops were grown in filtered, unpolluted air and in smoggy air. Research results show that a number of important California crops produce less yield, mature more slowly or suffer tissue damage when grown in smoggy conditions.

COTTON

California's $1 billion per year cotton crop, the state's biggest single farming product, is significantly affected by air pollution. Several varieties have been studied during the last decade to determine if and how air pollution cuts this crop's yield.

In addition to obvious leaf markings, ozone causes the flowers to drop off resulting in fewer bolls per plant. A study of the SJ-2 variety of cotton, the most common variety grown in the San Joaquin Valley, showed a 14 percent loss in weight of fiber length and elasticity which make cotton stronger and more marketable.

Other cotton varieties show differing degrees of sensitivity to ozone. Through the use of computer models, scientists estimate that the average cotton yield loss from ozone during 1988 was about 16 percent, with the highest reductions estimated at about 44 percent in the southern San Joaquin Valley.

GRAPES

Research conducted by the University of California found that Thompson Seedless Grapes produced 25 percent less yield in the San Joaquin Valley due to air pollution. Grapes grown in clean air had larger bunches and more of them.

Another study using Zinfandel grapes, however, showed that this variety formed less sugar when grown in polluted conditions. Zinfandel grapes grown in smoggy Riverside produced 60 percent less yield. Statewide, the average loss estimate for all grapes was approximately 25 percent during 1988.

Air pollution can reduce the size and weight of Thompson Seedless grapes, one of the San Joaquin Valley's most popular varieties, by up to 25 percent.

POTATOES

Russet "Centennial" potatoes, a valuable crop in the San Joaquin Valley, is particularly susceptible to air pollution damage. A study done in Riverside, California demonstrated losses in yield of more than 40 percent in total potato number and yield in smoggy air. Both leaf and root dry weights are also reduced.

BEANS

The leaves of kidney beans and most other beans develop dead and yellow spots in smoggy air and the plants die sooner than those grown in clean air. Plants grown in clean air also begin flowering earlier and, as a result, set pods earlier. Even more important, the effects of ozone and sulfur dioxide reduce the weight, number of seeds and pods and yield of kidney beans. Other beans are expected to show similar effects.

Air pollution can cause discoloration of leafy plants such as lettuce, causing them to lose salability.

LETTUCE

Lettuce, when exposed to polluted air, produces smaller, lighter heads. More importantly, leaves develop dead areas which are critical to their market value. Losses in yield are seen even when there is little or no leaf damage. Additionally, the exposed lettuce is thin and fragile to the touch, suggesting that it might suffer more damage in transit to the grocery store. These effects occur at ozone levels below the current California air quality standard (0.09) parts per million parts of air for one hour.

RANGE AND FORAGE GRASSES

Both total yield and quality of forage and range grasses are affected by air pollution which could have serious consequences for the state's livestock industry. Compared to grasses grown in clean air, loss in dry yield of grasses grown in smoggy air is as high as 10–20 percent. Additionally, ozone reduces carbohydrate levels of grasses by up to 56 percent.

NATIVE PLANTS

Air pollution is known to harm all major native plant groups, including flowering plants, conifers, ferns, mosses, lichens and fungi. In the Geysers region of Napa, Lake and Sonoma counties, injury to native plants, such as oaks and maples, has taken place downwind of geothermal power plants. Trees and other plant life in the San Joaquin Valley and adjacent Sierra Nevada suffer from air pollution generated in the urban areas. In addition, trees in the Sequoia-Kings Canyon National Park and the Sequoia and Sierra National forests have been injured by smog ormed in the San Joaquin Valley.

Since vegetation injured by air pollution was first noted in Southern California, it is not surprising that the national forests in the South Coast Air Basin continue to show moderate to heavy injury. Pine needles exposed to ozone develop yellow, blotchy marks and needles older than two years fall off, giving branches a scraffly, whiskbroom appearance. Needles and debris from trees killed by smog not only increase the risk of forest fire, but reduce seed germination and the chances of seedling survival.

Coastal sage scrub, chaparral, and native plants in the Mojave Desert are also sensitive to air pollutants. The most important effect is a reduced ability to cope with drought, disease and insects. Air pollution may put these plants at a reproductive disadvantage by causing them to produce fewer seeds. These conditions can lead to changes in succession resulting in a totally different plant community occupying a site.

ALFALFA

Air pollution also reduces the yield of alfalfa grown in the San Joaquin Valley. A study performed by the University of California found that current levels of air pollution reduce the yield of Moapa, a variety of alfalfa, by eight percent. The study also shows that relatively low levels of sulfur dioxide reduce Moapa yield by ten percent. Another important aspect of alfalfa production is how long a planting lasts in the field. This study, which was carried out over three years and involved 20 cuttings of the alfalfa, showed that smoggy air reduces hardiness and persistence, allowing weeds to invade the fields, and reducing net income per acre. Leafiness, the amount of leaves vs. the amount of stems, is also reduced in the Moapa variety.

ESTIMATED PERCENTAGES OF CROP YIELD LOSSES FROM OZONE DURING 1987–88

	1987 Mean	Range	1988 Mean	Range
Alfalfa	8.4	1.4–11.0	8.8	1.3–11.1
Beans	10.1	1.9–13.1	10.6	1.7–13.3
Cotton	20.9	15.8–31.7	16.4	6.2–44.9
Grapes	28.5	25.7–31.3	27.8	25.2–30.7
Oranges	20.5	7.2–40.0	32.0	11.3–63.3
Potatoes	14.7	9.9–19.4	15.2	10.3–20.1
Rice	5.7	3.2–7.2	5.0	2.7–6.5
Tomatoes (processing)	3.9	1.6–9.2	3.8	1.7–8.5

Summer season crops, such as these, suffer more damage from air pollution than winter crops due to greater air pollution levels.

Special care is given to assure that factors other than air pollution do not harm plants being studied.

CITRUS FRUITS

In a study of the effect of air pollution on commercial citrus trees, navel orange trees produced approximately 50 percent more fruit when protected from smog. Also fewer leaves were dropped by trees protected from smog. The statewide average yield loss for citrus was approximately 11 percent for 1988.

TOMATOES

Both ozone and sulfur dioxide can reduce the yield of canning tomatoes. Growing tomato plants in the outside air of Riverside, where some of the state's highest ozone levels are recorded, reduced commercial yield, plant weight and the number of red tomatoes. Pulp color also was below acceptable canning standards. Because of these losses in quality, tomatoes are among the cash crops no longer grown in metropolitan Southern California.

ORNAMENTAL PLANTS

Many types of shrubs, annual flowers, lawn grasses, trees and other plants grown in urban areas are sensitive to air pollution. Disfigured leaves and fewer blossoms can detract from the beauty and value of the ornamental plants that Californians spend millions of dollars for each year.

COMMON ORNAMENTAL PLANTS SENSITIVE TO SMOG

Zinnia	Oleander
Sycamore	Lilac
Petunia	White Birch
Fuschia	Rose
Periwinkle	Primrose
Azalea	Blue Grass

OS ANGELES ARBORETUM:

Air pollution research came full circle when the Air Resources Board began growing flowers and shrubbery in Southern California at the Los Angeles County Arboretum.

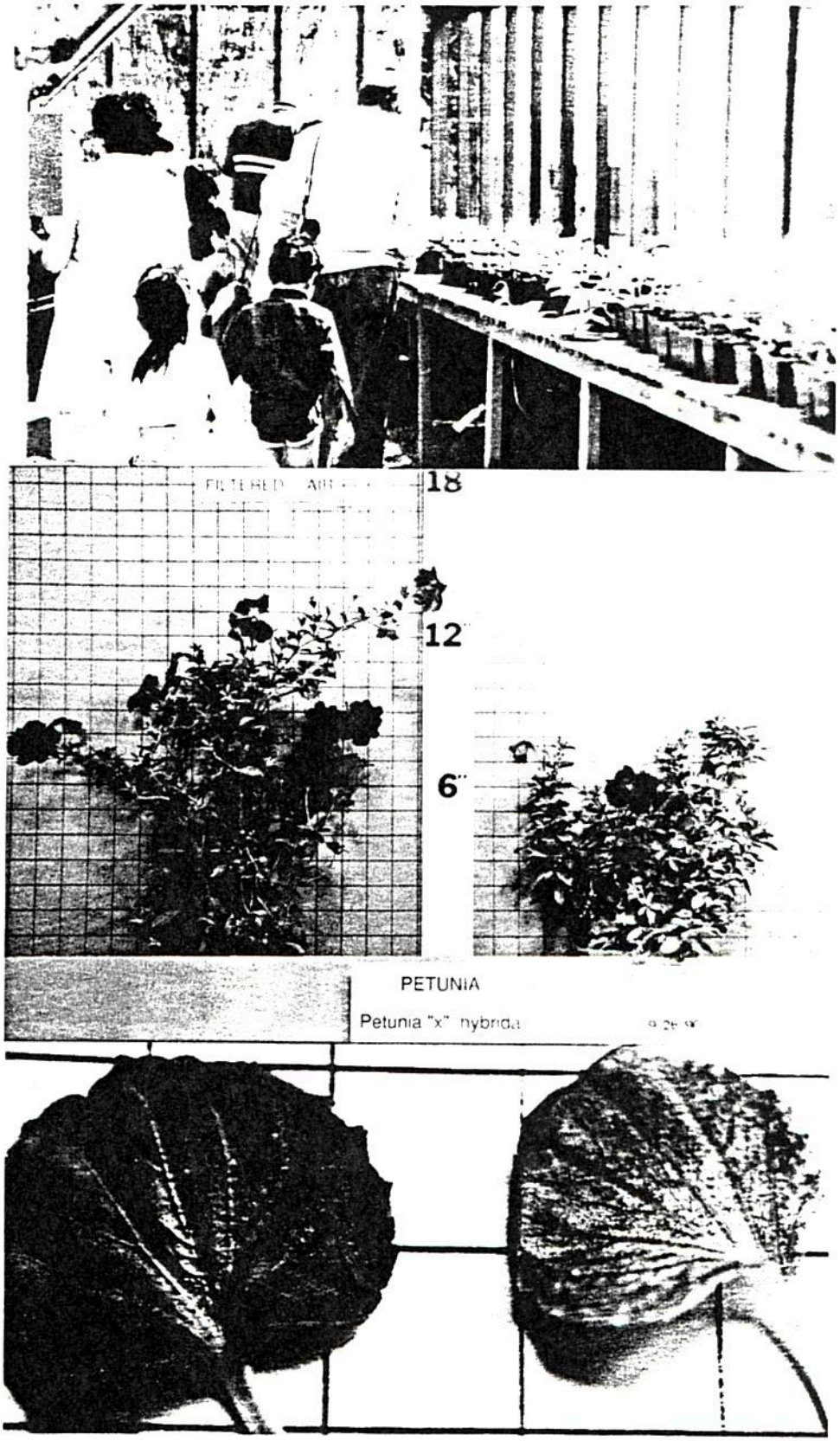
Dr. Arie Haagen-Smit, the ARB's first chairman, and the "Father" of smog research in California, began studying smog damage to plants after observing problems in his own backyard gardens in the late 1940's.

Haagen-Smit, a Biochemistry professor at the California Institute of Technology in Pasadena and a former plant researcher at the University of Utrecht, in the Netherlands, couldn't explain the damage to flowers in his garden. After ruling out other causes such as mineral and insect damage, he began to wonder if the brownish haze over Los Angeles might be causing his problems.

The rest is history. Haagen-Smit ent on to become one of the world's oremost authorities on air pollution science and, in 1968, the Chairman of the newly-formed California Air Resources Board.

The public education project exhibits identical varieties of smog-sensitive flowers and shrubbery in adjoining greenhouses. The plants receive the same amount of water, nutrients and care. The greenhouses however, get their air from different sources. One receives ambient Los Angeles air, while the other's air supply is filtered to remove impurities, including air pollution.

The experiment, designed to show the difference poor air quality can make in the health of common ornamental plants, has proven its point. The plants grown in the cleaner, filtered air are taller, fuller, and healthier than the same species grown in the greenhouse using mbient air.



Plants grown in clean filtered air are larger and healthier than those grown in polluted Los Angeles air at the ARB's Los Angeles County Arboretum exhibit.

WORK IN PROGRESS

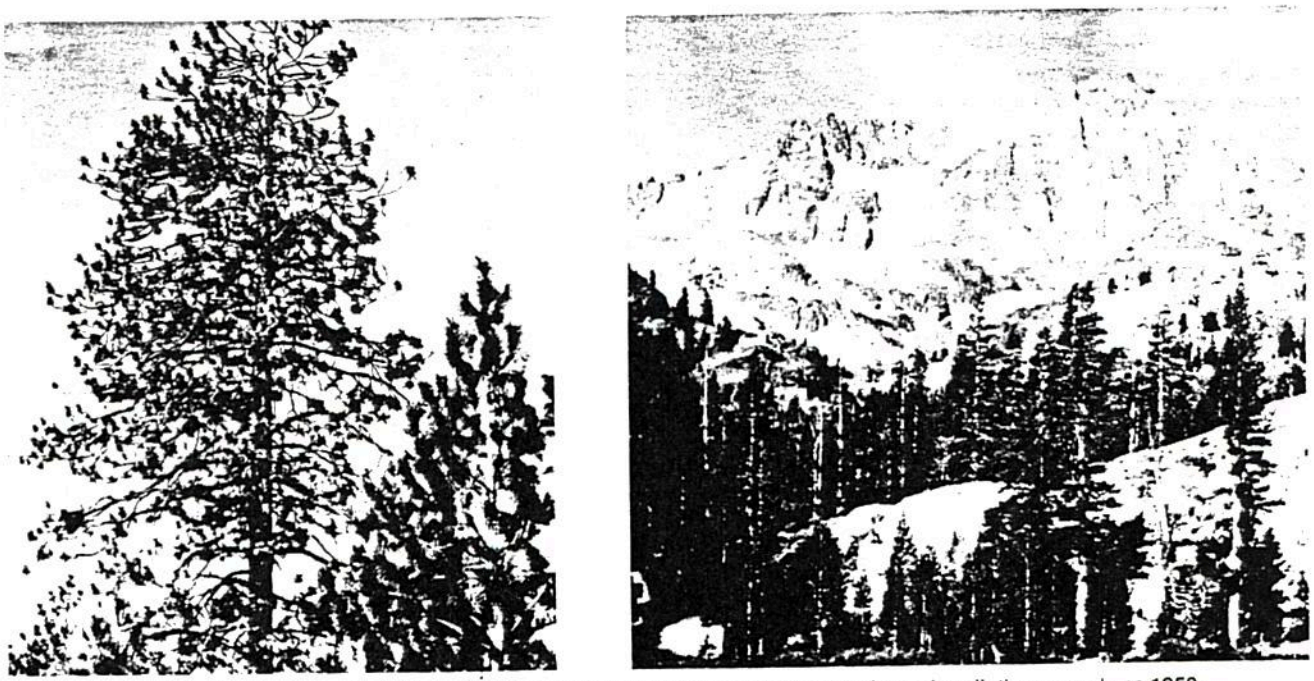
Forests

A multi-year study to determine how ambient air pollution affects seedling and young trees is nearing completion in the mountains above Bakersfield. In addition, a joint project between the ARB and the United States Forest Service is recording ozone exposures and signs of tree injury in forests on the Sierra Nevada's western slopes from Lake Tahoe to Fresno. Future research on those forests is expected to include long-term monitoring to better determine if changes occur following repeated exposures to ambient levels of ozone and acid deposition.

Tree Crops

The ARB is continuing to conduct research to determine the effects of air pollution on tree fruits and nuts. Research is currently being conducted to determine smog's effects on plum trees. Early results show that air pollution may cut plum yields and may affect tree vitality. It is hoped results of this project will also provide valuable information about other stone fruits.

Other ARB research includes the Crop Loss Assessment Program, in which scientists are developing techniques to combine the effects of smog on individual crops with regional air pollution levels to better anticipate economic losses caused by air pollution. The project emphasizes economic damage in the San Joaquin Valley where 90 percent of the state's agriculture is grown.



Mountain forests downwind of Los Angeles showed some of the first damage from air pollution as early as 1950.

Further Reading

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California Statewide Assessment of the Effects of Ozone on Crop Productivity, D. M. Olszyk, H. Cabrera, and C. R. Thompson, J. Air Pollution Control Assoc., 1988.

Crop Loss Assessment for California: Modeling Losses with Different Ozone Standard Scenarios, D. M. Olszyk, C. R. Thompson, and M. P. Poe, Environ Pollution, 1988.

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Protection for Sensitive Populations

Current law requires the primary standards to be set at a level which protects not only the general public, but sensitive groups within the population, such as bronchial asthmatics and emphysematics. The law does not, however, specify how large a sensitive group must be, and there have been complaints that the requirement leads to extraordinary expenditures to protect tiny fractions of the total population.

The Senate report accompanying the 1970 amendments offered the following guidance to the Administrator to help determine the pollution level which is protective of public health:

> *Ambient air quality is sufficient to protect the health of such persons whenever there is an absence of adverse effect on the health of a statistically related sample of persons in sensitive groups from exposure to the ambient air. An ambient air quality standard, therefore, should be the maximum permissible ambient air level of an air pollution agent or class of such agents (related to a period of time) which will protect the health of any group of the population.*
>
> *For purposes of this description, a statistically related sample is the number of persons necessary to test in order to detect a deviation in the health of any person within such sensitive group which is attributable to the condition of the ambient air.*

The secondary standard is to be set at a level "requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such pollutant."

The sensitive populations protected are on the following page.[104]

The Roles of EPA, CASAC and the SAB

The process for establishing national ambient air quality standards is lengthy, complex, inherently difficult and almost invariably controversial.

The process for establishing national ambient air quality standards is lengthy, complex, inherently difficult and almost invariably controversial.

The process is lengthy because of the need to translate sometimes voluminous—but always evolving—scientific data into a judgment of what levels of pollution jeopardize health. It is complex because the data ranges from studies on single species of laboratory animals to massive studies of tens of thousands of humans. It is inherently difficult because scientists, while able to identify biochemical or physiological changes caused by exposure to a pollutant, are almost always limited by their ability to state unequivocally whether a specific change is—or is not—an adverse human health effect. It is almost invariably controversial because no matter how lenient the standard may be, there will always be industries faced with the prospect of having to spend money in order to comply, and no matter how stringent, there will always be large numbers of Americans who will continue to suffer, because the Clean Air Act protects only sensitive groups, not everybody.

Setting a standard thus is not so much a matter of reaching a scientific conclusion as making a judgment—based on scientific facts, estimates, and hypotheses drawn from still emerging data—of what regulatory action is needed to prevent harm to human health.

Although the primary responsibility for setting the standards lies with the Administrator, the law requires the establishment of a seven-member committee to provide advice and recommendations. This group, known as the Clean Air Scientific Advisory Committee (often referred to as CASAC),

Sensitive Populations[a]

Pollutant	Sensitive Population	Percentage of Total U.S. Population	Number of Persons in Sensitive Population
Ozone	• Those with respiratory disease • Elderly • Pre-adolescents • Those exercising (e.g. jogging) • "Responders" (5 to 20 percent of the "normal" population)[b]	• 5.1–11.2 percent[c] • 12.7 percent • 20.6 percent • 4.7–23.8 percent[d,1] • 5 to 20 percent	• 13,820,000 • 32,284,000 • 52,517,000 • 10.8 to 54.6 million • 12.8 to 51.0 million
Sulfur dioxide	• Those with respiratory disease • Elderly • Pre-adolescents	• 5.1–11.2 percent[c] • 12.7 percent • 20.6 percent	• 13,820,000 • 32,284,000 • 52,517,000
Carbon monoxide	• Pregnant women • Those with ischemic coronary disease (e.g. angina)	• 1.6 percent • 2.8 percent	• 4,010,000 • 7,160,000
Lead	• Children under 5 • Pregnant women	• 7.6 percent • 1.6 percent	• 19,512,000 • 4,010,000
Particulate (PM10)	• Those with respiratory disease • Elderly • Pre-adolescents	• 5.1–11.2 percent[c] • 12.7 percent • 20.6 percent	• 13,820,000 • 32,284,000 • 52,517,000
Nitrogen dioxide	• Those with respiratory disease • Pre-adolescents	• 5.1–11.2 percent[c] • 20.6 percent	• 13,820,000 • 52,517,000

[a]This data reflects the latest information available at the time of publication and is based on based on *Vital Statistics: Current Estimates from the National Health Interview Survey, 1993*, Public Health Service, Centers for Disease Control, U.S. Department of Health and Human Services (1994) and other official surveys.

[b]Responders are individuals who, for reasons that are not fully understood, have more extreme reactions to ozone exposures than "normal" subjects. Although their reactions differ in severity from those of most subjects, they possibly represent one end of the normal distribution curve of reactivity to ozone. For purposes of this table and other charts, the lower end, or 5 percent, is used to represent responders.

[c]For purposes of this table, respiratory disease includes asthma, chronic bronchitis and emphysema. Information on the prevalence of these diseases is collected on the basis of interview surveys in which individuals report themselves as suffering from a particular disease. Because some individuals report themselves as suffering from more than one of these three respiratory diseases, merely adding the incidence rates for them would overstate the true prevalence. The reported incidence rates are asthma, 5.1 percent (13.1 million individuals); chronic bronchitis, 5.4 percent (13.8 million) and emphysema 0.74 percent (1.9 million). In this and other tables, although the full range of incidence is shown, the rate for chronic bronchitis is used for calculation of specific numbers.

[d]The number in this category varies according to eight different vigorous sports activities (basketball, bicycling, football, hiking, jogging, skiing, soccer, and tennis) or occupations (e.g. mail carriers). For exercise, the number ranges from 10.8 million joggers to 23.8 million bicyclists, or from 4.7 to 23.8 percent of the U.S. population. Although this table displays the full range of exercisers, for purposes of specific calculations the number of joggers, adjusted to eliminate double counting (e.g. so that asthmatics who are joggers are not counted twice), is used. See the endnotes for more information.

1

Activity	Number of Individuals	Percentage of Population
Basketball	28,181,000	12.3 percent
Bicycling	54,632,000	23.8 percent
Football	13,494,000	5.9 percent
Hiking	21,619,000	9.4 percent

Activity	Number of Individuals	Percentage of Population
Jogging	21,932,000	9.5 percent
Skiing	14,252,000	6.2 percent
Soccer	10,819,000	4.7 percent
Tennis	17,323,000	7.5 percent

must by law include at least one member of the National Academy of Sciences, one physician, and one person representing State air pollution control agencies. The mandated role of CASAC also requires that it—

> *. . . (i) advise the Administrator of areas in which additional knowledge is required to appraise the adequacy and basis of existing, new, or revised national ambient air quality standards, (ii) describe the research efforts necessary to provide the required information, (iii) advise the Administrator on the relative contribution to air pollution concentrations of natural as well as anthropogenic activity, and (iv) advise the Administrator of any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance of such national ambient air quality standards.*

Under current procedures, the Agency prepares a draft criteria document for review by the public, as well as CASAC. As the criteria document is being developed, a staff paper, which summarizes the evidence and conclusions in the criteria document and analyzes their significance, is also being prepared. These two are prepared by different offices at EPA: the criteria document by the Office of Research and Development; the staff paper by the staff of the Office of Air and Radiation.

The criteria document and staff paper are then revised based on CASAC review and public comment. These revised documents are forwarded for decision to the Administrator, who officially selects a proposed standard, which is published in the Federal Register as a proposal. After a public comment period, the proposed standard is reviewed and modified, as appropriate, and a final standard is published.

The time required for this process of identifying criteria pollutants, developing the supporting information and documents, and setting the standard can take several years, depending on the pollutant and whether the standard is new or a revision. Despite the massive amount of time and work devoted to setting standards—and the changes that have been made to assure that the process is open and objective, and that the science is thorough and reliable—criticism from polluters has been virtually unrelenting for the quarter century that the process has existed.

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Criticisms and Previous Responses

Before setting the standard, the studies that will form the data base must be conducted, collected, and analyzed. This process, as distinct from actually setting the standard, has been the subject of sharp and continuing criticism. At various times, Congress has acknowledged these complaints and either amended the law or taken other action.

In response to complaints in the early 1970s about the quality of the scientific studies underlying the standards, the Senate Committee on Environment and Public Works commissioned a two-year comprehensive review by the National Academy of Sciences-National Academy of Engineering. This task was defined during the opening remarks of the Conference on Health Effects of Air Pollution (October 3–5, 1973) as follows:

> *We have therefore asked the National Academy of Sciences to gather the best minds that it can find, to attempt to validate the information we have, to identify areas of certainty and uncertainty, to review the adequacy of margins of safety, to show areas where most research is needed, to show what is known and what is not, to identify the population groups we are protecting, to point out errors and doubts in data, and to come back to the Congress with its best judgment—in a preliminary form from this meeting and in a final form 10 months from now.*

When the Act was reviewed in 1976 and 1977, there were again complaints that the standards were based on outdated or faulty studies, and should be revised. In response to these criticisms, the 1977 amendments required the standards to be reviewed and, where

appropriate, revised. This was to be done by December 31, 1980, and thereafter at least once every five years. In response to complaints that the standards were based on poor scientific studies, the Congress established the seven-member Clean Air Scientific Advisory Committee. Every five years CASAC is to review the criteria published under section 108 and the standards promulgated under section 109 and recommend any revisions which it considers appropriate. The 1977 amendments also required EPA to publish, together with any proposed standard under sections 109, 111, or 112, CASAC's comments and the Agency's basis for the proposed standard.

In 1978 Congress again acknowledged criticisms of the Agency's scientific base by giving statutory recognition to the Science Advisory Board (SAB). This group had been created by the Agency in 1974. The 1978 amendment required the SAB to subject "any proposed criteria document, standard, limitation or regulation under the Clean Air Act" to a technical and scientific review. CASAC is the mechanism by which the SAB complies with this requirement.

With a staff of nearly sixty and after two-years of comprehensive evaluation of literally every provision of the law, the Commission recommended that—

The current statutory criteria and requirements for setting air quality standards at the levels necessary to protect public health without consideration of economic factors, should remain unchanged.

Complaints about the Clean Air Act generally caused the Congress to create a special 13-member National Commission on Air Quality charged with conducting a top-to-bottom review of the Clean Air Act. It included representatives of industry, labor, public interest groups, states, cities, tribes, as well as Members of Congress. Among these members were Rep. David Stockman, who later became first director of the Office of Management and Budget under President Reagan; Rep. John Dingell, later Chair (now Ranking Minority Member) of the Committee on Energy and Commerce of the House of Representatives; and, the senior Republican on that Committee at the time, Rep. James T. Broyhill. With a staff of nearly sixty and after two-years of comprehensive evaluation of literally every provision of the law, the Commission recommended that—

> *The current statutory criteria and requirements for setting air quality standards at the levels necessary to protect public health without consideration of economic factors, should remain unchanged.*[105]

During the 1980s, criticisms of the law's standards and standard-setting process became more muted, but persisted. In response, the committees of both the House of Representatives and the Senate conducted their own reviews of the Clean Air Act's provisions. Although there were two relatively conservative Presidents during this period, a House committee chaired by a frequently vocal critic of the law and a Senate Committee with senior members (and from 1987 to 1990, a Chairman) who were unenthusiastic about it, no changes in standards or standards-setting process were recommended or adopted.

Despite repeated evaluations of the Clean Air Act over the past quarter century and many attempts to respond to legitimate criticisms, some groups remain unsatisfied. This suggests that their complaint is not with the fairness and objectivity of the standard-setting process, but rather with its ultimate goal: protection of human health. There are some groups of polluters that continue to insist that standards should be based on cost to them, rather than avoiding injury to their victims. ***In considering criticisms of the process—including its***

Despite repeated evaluations of the Clean Air Act over the past quarter century and many attempts to respond to legitimate criticisms, some groups remain unsatisfied. This suggests that their complaint is not with the fairness and objectivity of the standard-setting process, but rather with its ultimate goal: protection of human health.

requirements for protecting sensitive groups and providing a margin of safety—it remains essential to bear in mind that critics sometimes have the undisclosed agenda of repealing the Act's health basis, not improving its science.

facts about

Air Pollution and Health

Air pollution is part of everyday life for millions of Californians in every urban region of the state. Residents are regularly exposed to air pollution levels that can cause nausea, headaches, dizziness, and shortness of breath, even among healthy adults.

Even though California administers the world's strictest air pollution control program—including trendsetting emission standards for motor vehicles as well as industrial facilities—the state also has the nation's highest air pollution levels.

Annual doses of unhealthy ozone, or urban smog, have been cut in half over the last 15 years in the South Coast Air Basin as a result of these strict standards. Nonetheless, air quality in that region continues to be the nation's worst. In other urban areas, the ARB's program has kept pace with the explosive growth that has made California the nation's most popular place to live.

Although air pollution may obscure visibility, the most important reason for regulating it is the health problems that it causes. Because of the unique combination of high pollution levels and the large number of people exposed to them, the potential health threat from smog in California is greater than that in the remaining 49 states combined.

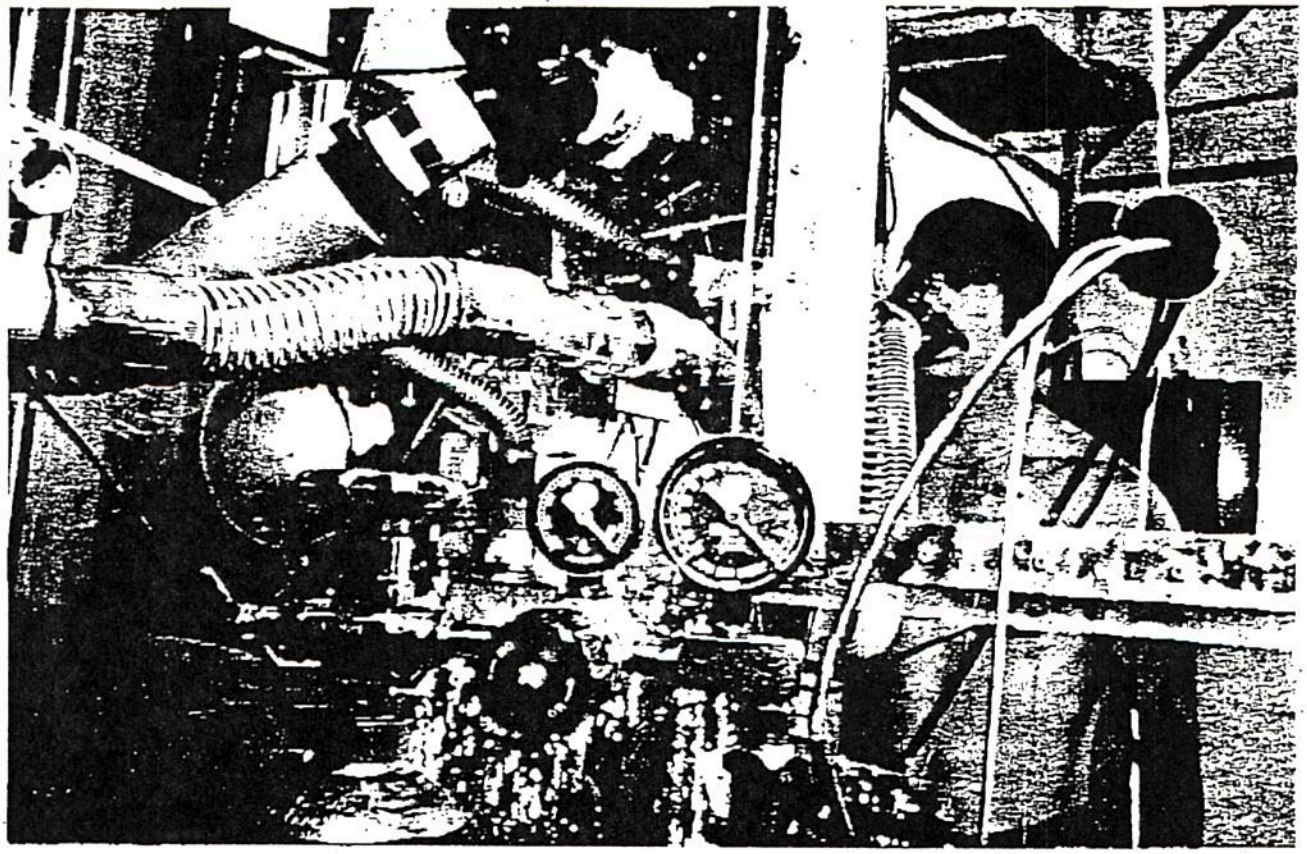
While air pollution affects everyone to one degree or another, some people are extremely susceptible to severe health damage. That includes young children whose respiratory systems are still developing; those who suffer from existing heart and respiratory diseases; and healthy adults who exercise vigorously.

These people represent a sizeable part of the population, up to one person in five by some estimates, equal to the population of the San Francisco Bay Area.

In some urban areas, ozone levels are high enough to trigger health advisories or smog alerts during the summer months. When these levels are reached, even healthy adults and children are advised to avoid or reschedule sustained strenuous outdoor exercise such as soccer and long-distance running. Individuals with heart or lung problems are further advised to reduce their activity and exposure.

To protect health, the Air Resources Board sets air quality standards which are based on research that documents harmful pollutant levels. California's air quality standards are stricter than those set by the federal EPA for the rest of the nation. Essentially, the state's definition of "healthy" air is based on lower pollution levels than those used nationally. In addition, because the state has to deal with pollution problems that are not prevalent elsewhere, California has adopted some standards for which there is no national counterpart.

The adoption of these unique standards reflects recent research findings that some pollutants—especially urban smog or ozone—are more harmful in lower concentrations than previous research suggested. That research also shows that high pollution levels can cause immediate health problems and that chronic exposure to lower concentrations may be the basis for life-long, permanent health damage.



ARB research has established that air pollution:

- aggravates cardiovascular and respiratory illnesses;
- adds stress to the cardiovascular system, forcing the heart and lungs to work harder in order to provide oxygen;
- speeds up the natural aging process of the lung, accelerating the loss of lung capacity;
- damages cells in the airways of the respiratory system;
- damages the lungs even after symptoms of minor irritation disappear;
- contributes to the development of diseases including bronchitis, emphysema, and possibly cancer.

AIR QUALITY STANDARDS
California's clean air goals

California's air quality standards are the state's definition of healthy air. In effect, they tell us how much of a substance can be in the air without causing harm.

In most cases, California's health standards are stricter than similar standards adopted by the EPA for the rest of the nation. They are the goals of the ARB's regulatory program, which reduces emissions to ultimately reduce pollutant concentrations to healthy levels.

California legislation requires the ultimate attainment of these unique, health-based standards and requires steady emission reductions until those goals are met.

How much? How long?

Air quality standards define allowable concentrations and the allowable duration of exposure. Concentrations are typically expressed in units such as "ppm" (parts of the substance for each million parts of air) or "ug/m3" (micrograms of the substance per cubic meter of air). Duration is the time period of concern, usually expressed in hours. The California air quality standard for ozone, for example, is 0.09 ppm (180 ug/m3) averaged over one hour.

Law and Science

California law requires that air quality standards be adopted "in consideration of the public health, safety, and welfare including, but not limited to, health, illness, irritation of the senses, aesthetic value, interference with visibility and effects on the economy."

Based on this legal requirement to protect health and welfare, standards are adopted after considering information from different types of scientific research.

Health standards are based on two primary types of research. They include epidemiology, which studies groups of people in their normal environment and laboratory studies, which can be of people or animals exposed to pollutants, always under carefully controlled conditions. Because both types of studies have inherent strengths and weaknesses, both are needed to provide the most reliable scientific basis for air quality standards.

Standards to protect public welfare are founded on other types of research, which study such diverse factors as odor detection or the economic costs of lost crops or damaged materials.

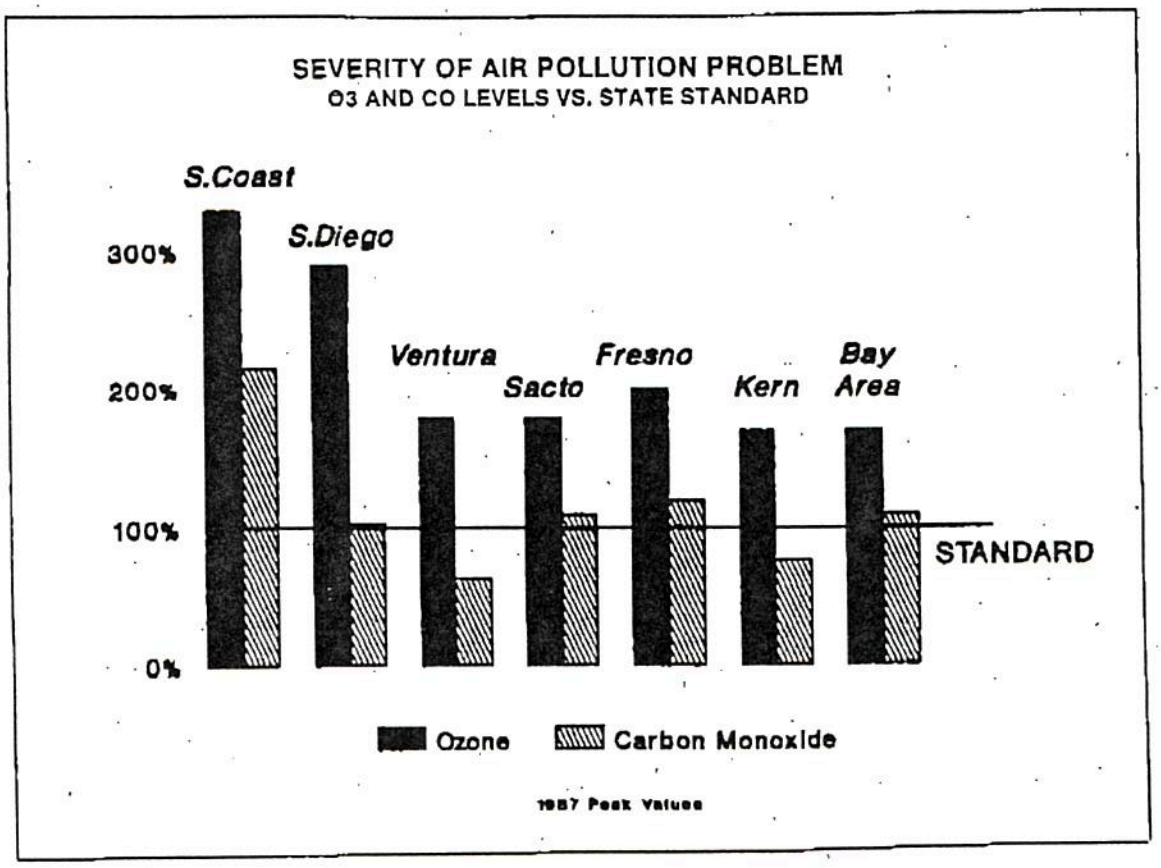
THE POLLUTANTS

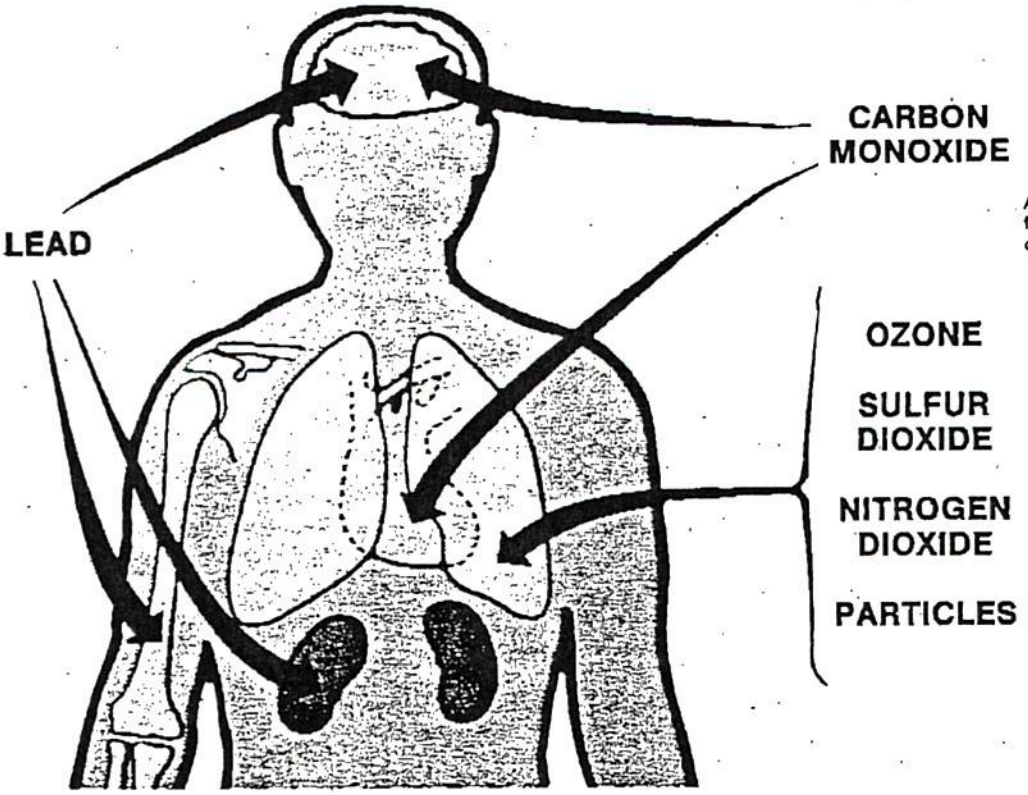
Some pollutants that pose health problems are directly emitted to the air. Others are formed in the atmosphere through chemical reactions among polluting gases that are triggered by sunlight.

OZONE is a colorless, odorless gas and the chief component of urban smog, it is by far the state's most persistent and widespread air quality problem. It is formed from the chemical reactions among hydrocarbons and nitrogen oxides.

Short-term exposure over an hour or two can add stress to the body. It is a strong irritant that can cause constriction of the airways, forcing the respiratory system to work harder in order to provide oxygen. Besides shortness of breath, it may aggravate or worsen existing respiratory diseases, such as emphysema, bronchitis and asthma.

Chronic exposure to ozone can damage deep portions of the lung, even after symptoms such as coughing or a sore throat disappear. Ozone can damage the alveoli, the individual





Air pollutants commonly found in California may affect different parts of the body.

air sacs in the lung where oxygen and carbon dioxide are exchanged. Over time, this membranous, filmy tissue is permanently damaged, reducing its ability to function and essentially accelerating the natural loss of lung capacity.

ARB research has provided preliminary evidence that some degree of permanent lung damage may occur in young adults, aged 14 to 25, who are thought to have been life-long residents of the highly polluted South Coast Air Basin. This pilot study examined the lungs of young accident and homicide victims. The exact air pollution exposure and health histories could not be obtained for most victims. However, the frequency of certain types of lung damage observed in this young population raises concerns regarding the health effects of long-term exposure to air pollution, including high levels of ozone, found in the South Coast Air Basin.

The broadest finding was some degree of Centriacinar Region Disease (CAR), chronic inflammations of the bronchial tubes. Nearly all of the lungs examined had some form of chronic bronchitis and 76 percent showed some degree of inflammation. In addition, about one-third of the subjects had some degree of chronic interstitial pneumonia, a form of the disease found deep within lung tissue.

Similar and related investigations are underway or planned which will provide further information regarding the risk to human health that may be associated with long-term exposure to air pollution.

This recent research, together with findings from other population studies and studies performed in laboratory animals, provides compelling evidence to dispel the belief that the respiratory system fully restores itself from exposures to ambient air pollution.

California's health standard for ozone is .09 parts per million (ppm) for one hour, in contrast to the EPA national standard of .12 ppm. Reducing all ozone concentrations to this level, to protect the health of the state's most vulnerable people, remains one of the premier goals of the ARB's air pollution control program.

ARB standards also require health advisories when one-hour ozone concentrations reach .15 ppm, almost double the state's health standard. These "smog alerts" provide warnings so that residents can take precautions to protect their health from excessive ozone levels, usually by avoiding strenuous exercise or outdoor exposures.

CARBON MONOXIDE is the byproduct of incomplete combustion, primarily from motor vehicle exhaust. The highest concentrations are found in areas with congested or high volumes of traffic and during the winter months.

The state's air quality standards are 20 ppm averaged over one hour and 9 ppm averaged over eight hours. The standards are designed to prevent chest pain in moderately exercising people who have heart problems, but other types of health damage can result from higher concentrations.

Carbon monoxide is readily absorbed into the body from the lungs, where it binds with hemoglobin, which reduces the ability of this protein to carry oxygen. The result is reduced oxygen reaching the heart, brain and other tissues. This can be critical for people with heart disease, chronic lung disease or anemia, as well as unborn children. Even healthy people who are exposed to excessive carbon monoxide can experience headaches, fatigue, slow reflexes and dizziness.

Health damage caused by carbon monoxide is of greater concern at high elevations where the air is less dense, aggravating the consequences of a reduced oxygen supply. In consideration of this, the ARB has a special CO standard of 6 ppm averaged over eight hours for the 6,000 foot elevation, Lake Tahoe basin.

SULFUR DIOXIDE is produced primarily by the combustion of coal, fuel oil and diesel fuel. California's standards are 0.05 ppm averaged over 24 hours and 0.25 ppm averaged over one hour.

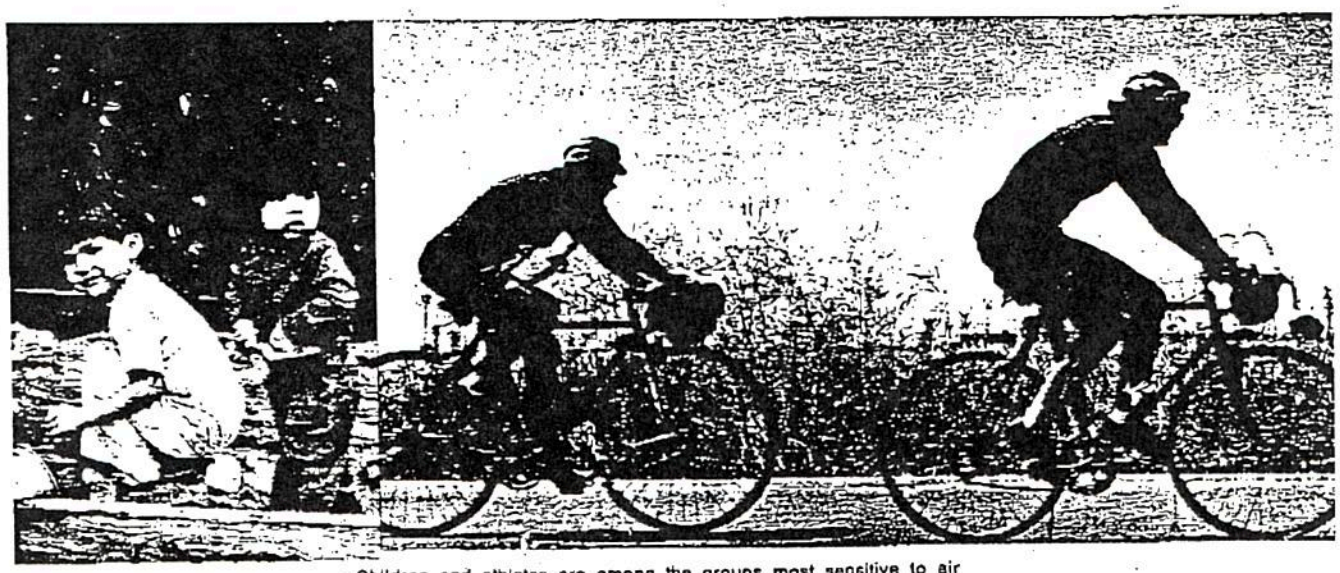
Sulfur dioxide causes a constriction of the airways and poses a particular health hazard for asthmatics. The air quality standard was set to protect them from breathing difficulties during and after short periods of exercise.

Children exposed to sulfur dioxide experience increased respiratory tract infections and healthy people may experience sore throats, coughing and breathing difficulties when exposed to high sulfur dioxide concentrations.

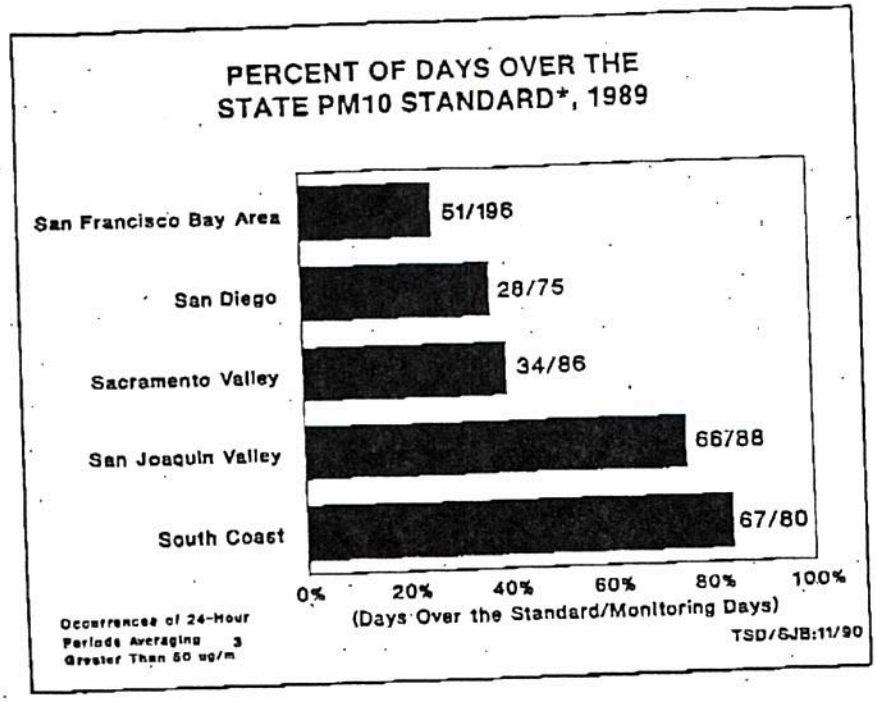
NITROGEN DIOXIDE is a byproduct of all combustion and is emitted from sources such as motor vehicles, industrial boilers and heaters. It is one of the pollutants known generically as nitrogen oxides, which are a major component of urban smog (ozone) and is responsible for its reddish-brown haze. In winter months, however, when the photochemistry that forms ozone is lowest, nitrogen dioxide concentrations remain high.

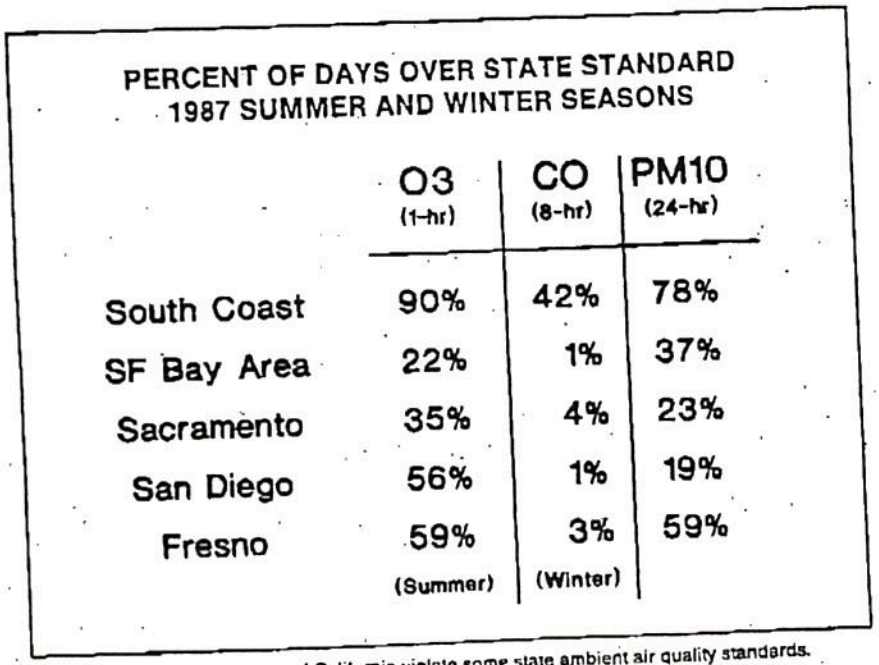
It is an irritating gas that may increase the susceptibility to infection and may constrict the airways of asthmatics.

The California air quality standard for nitrogen dioxide is 0.25 ppm, averaged over one hour.



Children and athletes are among the groups most sensitive to air pollution because they work harder or spend more time outdoors than average Californians.





Nearly all urban areas of California violate some state ambient air quality standards.

PARTICULATE MATTER can be emitted directly into the air, such as the case with diesel soot, wood burning or the result of agricultural operations. It can also be produced through photochemical reactions among polluting gases, primarily sulfur oxides and nitrogen oxides, resulting in corrosive sulfate or nitrate particles.

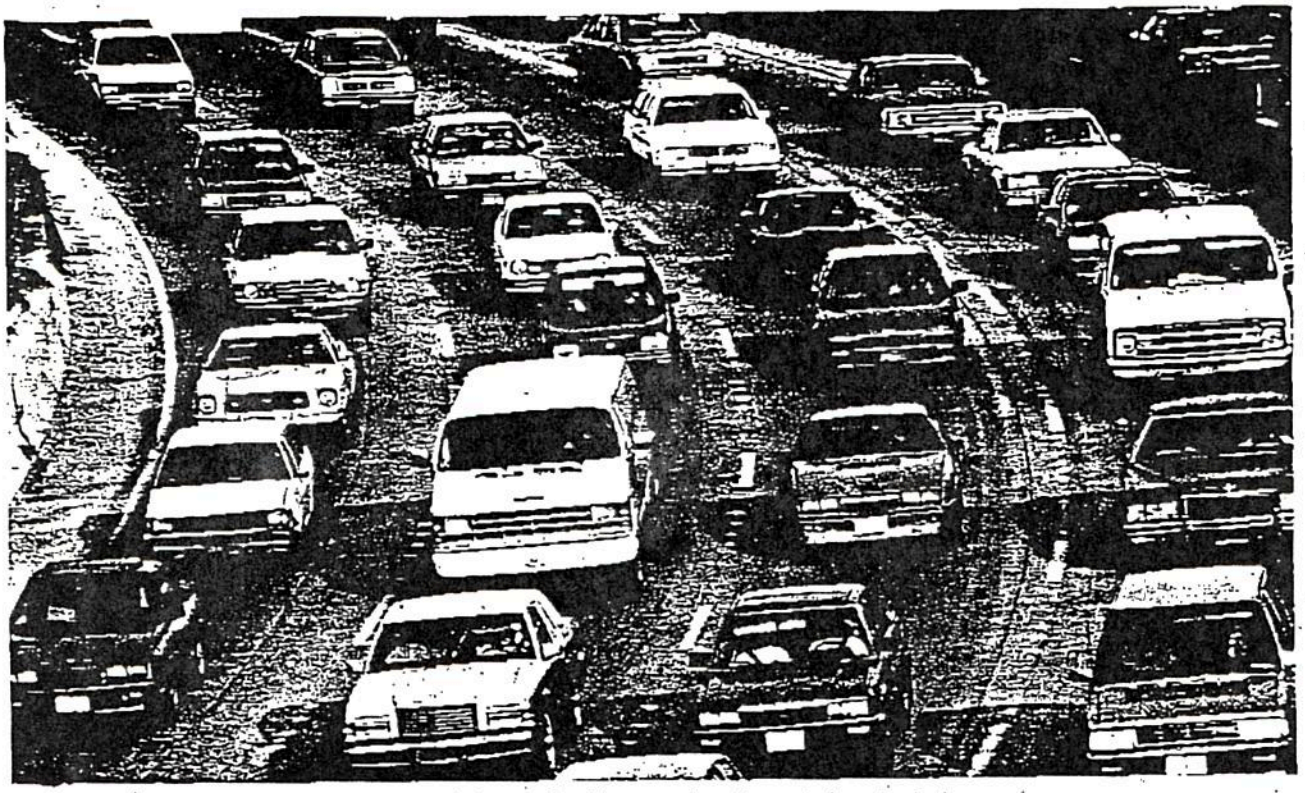
Although all particles can pose a potential health problem, the greatest concern is for microscopic, invisible particles which are the greatest health threat. These particles are less than 10 microns in diameter, about one-fifth the size of a human hair, and are known as PM-10.

The state's standards for these small particles are 30 ug/m3 averaged over a year and 50 ug/m3 averaged over 24 hours.

Concern for these particles is based on their ability to bypass the body's natural filtering system, posing a threat to the respiratory tract.

Short term exposures can lead to coughing and minor throat irritation. Longer term exposures can lead to increased bronchial disease. In addition, some of the directly emitted particulate, such as diesel soot and wood smoke, can be carriers for other toxic compounds including benzene and dioxin, increasing potential cancer risks.

Even though all particles ten microns or less are a health problem, they affect different parts of the respiratory tract depending on their size. Particles from 2.5 to 10 microns in diameter tend to collect in the upper portion of the respiratory system, affecting the bronchial tubes, nose and throat. Those particles 2.5 microns and smaller in diameter can infiltrate deeper portions of the lung and remain there longer, increasing the risks of long-term disease.



Because of the state's stringent automotive emission standards violations of both lead and carbon monoxide have been sharply reduced.

LEAD

At least 70 percent of the lead in the air is due to industrial sources. Airborne lead from automobile exhaust has been dramatically reduced in recent years, a direct result of lead reduction in gasoline required by ARB and EPA regulations.

Lead particles small enough to be inhaled into the lungs are readily absorbed into the blood and circulated throughout the body. The most important target of lead is the brain. At relatively low levels, lead exposure can result in a permanent decrease in the IQ of children. At higher levels, anemia can occur in both adults and children.

To protect public health, the California air quality standard for lead is set at 1.5 ug/m3 averaged over 30 days.

INDOOR AIR POLLUTION

The primary purpose of the ARB's program is to reduce outdoor pollution. But Californians are no different from people in other regions, spending an average of 80 percent of their time indoors, where they are also exposed to chemicals and pollutants.

The ARB has increased its research to study indoor pollution levels, prompted by concern that exposures are significant and can affect how people react to outdoor air quality. In addition, even when levels of indoor pollutants are low, protracted exposure to them can cause a significant health risk over a lifetime.

Indoor pollution can be generated by everyday activity, including cooking or the use of common household products, such as cleaning agents, paints and hairspray. In addition, common building materials and home furnishings can be a source of toxic vapors.

Recent advances in miniature, portable monitoring equipment have enabled the ARB to design research projects that measure the total amount of many chemical compounds that people are exposed to in a typical day.

Combined with studies that document activity and the sources of pollution to which people are exposed, the ARB will be better able to estimate the total amount of pollution that people breathe, and to develop more effective approaches to reducing total exposures.

Further Reading

Toxicology, Casarett and Doull's Toxicology, edited by Klassen, Ander and Doull, McMillan Publishing Co., 1986.

Ozone, Smog, and You, USEPA, Washington, DC 20460, September 1986.

Effects of Ozone on Health, Technical Support Document, Air Resources Board, September 1987.

Playing Safe on Smoggy Days, South Coast Air Quality Management District, El Monte, CA, October 1990.

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INTER-OFFICE CORRESPONDENCE

AAA MID-ATLANTIC

and ASSOCIATED COMPANIES

FOUNDED 1900

DATE: May 6, 1996

TO: Stakeholders Group

FROM: Jack E. Weber Jr.

Information contained in the attached Kay H. Jones' article entitled "*The American Lung Association is Blowing Smoke*" and the article by John W. Merline entitled, "*Urban Smog: How Bad a Health Hazard?*"should be considered a balance to the following previously distributed articles entitled: *"96 Ozone"*; *"Snapshot of Six Air Pollutants"*; and *"Questions and Answers About the Clean Air Act: Exploding Some Myths.*"

JW

JW:rmd
Attachments

THE AMERICAN LUNG ASSOCIATION IS BLOWING SMOKE

By Kay H. Jones, Ph.D.
Zephyr Consulting
Seattle, Washington

January 16, 1996

The American Lung Association's (ALA) recent report "Out of Breath" (Nov. 1995) represents either a dishonest or a naive portrayal of the current U.S. population's exposure to urban smog. The ALA claims that 161 million people live in areas that exceed .07 ppm ozone levels and are "*potentially*" exposed to "*unhealthful*" ozone levels. "Potentially" to the ALA is a broad cover your tracks caveat because the Association won't discuss the probability or likelihood of its claim which is zero! First of all, a .07 ppm, one exceedance per year standard is not being considered by EPA. The August 1995 EPA staff paper presented .07 to .09 ppm as a concentration range for consideration. They never focused on a one exceedance option but recommended a range of one to five exceedances while clearly stating that "there is little difference in health risk within the range..." The ALA 161 million person exposure estimate is based on juvenile accounting methods. The ALA assumes that only one monitor in a county represents the exposure for all persons in that county and further assumes that these same persons are standing outdoors next to that monitor 24 hours a day. Such over simplified assumptions might be excusable if the ALA were working in a vacuum, but it is not. The ALA has a designated representative who has been exposed to all of EPA's background documents on human exposure estimates and all of the EPA Clean Air Scientific Advisory Committee's (CASAC) review activities. CASAC makes the final recommendation on standards to the EPA Administrator. None of the ALA population exposure claims are supported by the very documents which it cites only in part and inaccurately. For example, the Nine City Study upon which susceptible population exposure estimates have been made for the various ambient standard options show that the variation between the low and high maximum annual one hour concentration varies by a range of 18-60% with an average of 34%. The ALA has 1993/94/95 data in hand

showing similar variation in all urban areas across the nation. Such deliberate oversight is inexcusable.

In using its naive model, the ALA suggests that 62.3% of all children in the U.S. between the ages of 5 and 13 are exposed to "unhealthful" levels of ozone above .07 ppm. Their estimates are 16.6% and 23.9% for a .09 ppm standard and the current 0.12 ppm standard respectively. Again, the ALA has deliberately ignored the truth. EPA has identified children who engage in moderate exercise outdoors as being the most susceptible population at risk. When this population is examined more closely in terms of its probable exposure and activity patterns, the residual risk is essentially indistinguishable among the three alternatives across the 9 representative cities studied by EPA. The range of the residual risk for the most sensitive effect in percent of outdoor children is 1.7-5.1% for a .07 ppm standard 5.2-14.3% for a .09 ppm standard, and 4.6-13.7% for the current standard. CASAC concluded that there is no "bright line" which distinguishes any of the proposed standards (either the ppm level or the number of exceedances) as being significantly more protective of public health. The ALA's numbers and conclusions are far from this scientific consensus.

The ALA also highlights supposed ozone impacts on the health of asthmatics ranging from 3 to 8 million individuals. Again, a more detailed EPA analysis contained in the staff paper and known to the ALA is totally ignored. A specific study has been conducted which shows a possible association between increased hospital admissions for asthma patients with increasing ambient ozone levels in New York City. The normal hospital admissions rate is approximately 28,500 asthmatics per year, unrelated to ozone. The past 3 year average ozone related added admissions is estimated to be 225 per year or 0.79% above normal. The incremental increase estimates after achieving the two background standard options discussed by the ALA are 60 additional admissions for the for the .07 ppm option and 180 for the .09 ppm option. The difference is only 0.42%. For the ALA to in any way infer that this is a significant differential impact is absurd.

In its press release the ALA attacks the .09 ppm 5 exceedance option as being too lax because this standard could be exceeded 15 times in 3 years suggesting that such exposures are unacceptable. This is also contrary to CASAC's conclusion about the choice among the various standard options. The EPA rationale for allowing multiple exceedances is to prevent the administrative chaos which occurs when an area that has achieved attainment status bumps marginally back into "non-attainment" due to extreme meteorological conditions. This in fact occurred in 7 cities in 1995 because of the current one exceedance per year constraint. Why has the ALA taken such a strident position on wanting a .07 ppm, one exceedance standard

in light of the preferences of the CASAC expert panel? Of the 10 panel members 8 enumerated their personal preference. None favored the .07 ppm option. Three favored a .08 ppm threshold. Three favored .09 ppm. One favored .08 to .09 and one favored .09 to .10 ppm.

Why is the ALA so far out in left field on the ozone issue? Contrary to its exaggerated portrayal of the current ozone problem, there are less than 20 cities in the U.S. outside of California which would be classified non-attainment, based on current data. Only Houston would be above the marginal and moderate classifications. Changing the current standard to .08 ppm or .09 ppm will not alter the future non-attainment picture from a regulatory perspective. There is no real reason to change the current standard from a public health protection perspective. The only reason for doing so relates to aligning the exposure period with the effects data base as well as reducing the risk of bumping up into marginal non-attainment .

The ALA attempts to project a medical science based image to the public. It is unfortunate that its previous reports on air pollution exposure and the one reviewed here, "Out of Breath," are just so much propaganda. If the ALA is seeking credibility it needs to embrace the tenet that sound environmental protection policy can only be achieved through the application of sound science.

Zephyr Consulting

Urban Ozone Population Exposure Fact Sheet

by Kay H. Jones, Ph.D.

Statement:

President Clinton recently stated that 90 million or 1/3 of the Nation's population is breathing unhealthy air.

Facts:

1. The President has been misinformed by his data sources. EPA has reported that there are 31 "non-attainment" urban areas in the U.S. based on 1992/93/94 monitoring data. 24 of these urban areas are outside of California. A non-attainment designation means that at least one monitor in the region recorded 4 or more days above the standard during the 1992/93/94 three year measuring period. (If a monitor showed only 3 days above the standard in three years, the region would not be classified as "unhealthy" according to Clinton.) The President was citing the total population living in the 31 urban areas, instead of what the actual exposure is, which is a huge misrepresentation.

2. There are 117 counties that make up the 24 non California non-attainment areas which have ozone monitoring data.

3. Only 44 of the 117 counties showed violations of the ozone standard during 92/93/94, i.e., 4 or more days above the standard in the three year period.

4. Assuming that total county populations are exposed to the worst case monitoring data (which is not the general case) 21 counties showed only one excess exposure day above the standard in a three year period, 10 showed 2 days, 12 showed 3 days and Harris Co (Houston) showed 10 days.

5. California, in particular Southern California, is the extreme case, totally different from the rest of the Nation. The attached chart shows the distinct contrast.

6. The total exposure estimate is 1/2 of the Clinton claim. His claim relative to the total National population is distorted by the Southern California contribution.

7. If we examine the total U.S. exposure in terms of person days per year, only 5.4% of the total exposure occurs outside of California, of which 2% relates to Houston alone. 94.6% of the Nationwide exposure is in California.

8. If we assume that exposures greater than one day per year are considered unhealthy (which is not the case) the non- California portion of Clinton's 90 million person, i.e., 69 million are so exposed only 0.3% of a year. For the 23 million exposed in California they are so exposed 16.3% of the year. This is 54 times higher than the rest of the Nation.

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Little Threat to Most Americans

Urban Smog: How Bad A Health Hazard?

John W. Merline

This summer, like most, has brought its share of smog alerts: hot summer days when people are warned that air pollution, mainly smog, makes working or playing outside a possible health threat.

Over the past 20 years, the federal government has engaged in an all-out campaign to cut the amount of smog. It has set up standards that all areas must meet, and has imposed strict requirements on those failing to comply. In 1990, the federal government upped the ante. Communities failing the clean air test face even more onerous regulatory mandates from Washington—from centralized car emissions tests to industrial controls—costing billions of dollars. Still, the Environmental Protection Agency (EPA) says that almost one half of Americans may breathe unhealthy air—unhealthy by federal standards, that is. From the constant warnings about it, and the apparent willingness to spend enormous sums of money combating it, one would assume that smog is a rather lethal air pollutant to a sizable population.

But how threatening is it? The extensive medical research on the subject suggests that, for the majority of people exposed, smog is at worst a mild irritant, the effects of which are completely reversible. And most Americans have little to worry about; they live in areas where days with smog levels high enough to cause any symptoms are a relative rarity.

Smog is comprised principally of ground-level ozone, which forms from a complex photochemical reaction when nitrogen oxide and volatile organic compounds combine in the presence of sunlight and heat. (It is distinguished from stratospheric ozone, which makes up the Earth's protective ozone layer.) While cars, trucks, factories, and the like produce many of these ozone "precursors," natural vegetation produces volatile organic compounds as well. And, because sunlight and temperature stimulate the reaction, the highest levels of ozone (and smog) tend to form on hot, stagnant, summer days. For this reason, there is an "ozone season," typically the three months of summer. Almost all the smog problems occur in this climatic window.

There is no question ozone, a highly reactive substance, can be quite harmful at high concentrations. However, in this case, the question is how harmful is it at levels found in the air. For years, scientists have studied the effects of ozone on human health, looking both for short-term acute effects and long-term, or chronic, health problems. The concern is that urban smog is a serious health threat, both in the short and long term.

What have scientists learned? There is clear evidence that ozone pollution can cause some short-term breathing problems. For the most part, these involve coughing, shortness of breath, or pain on deep breathing. Some people, however, are especially sensitive to ozone, including asthmatics or those with other breathing problems.

Short-Term Effects. For healthy people, feeling the effect of ozone involves three factors. The first is the amount of ozone in the air. Ozone is typically measured in parts per million. According to the current federal standard, the average peak ozone level in a particular area shouldn't exceed 0.12 parts per million (ppm) on more than one day a year.

The second health factor is the length of exposure. A very short exposure, even to relatively high levels of ozone, probably won't produce symptoms. But longer exposures can.

The third factor is how deeply a person is breathing. Someone exercising vigorously, for example, would increase the dose of ozone into his lungs. Studies have shown that people relaxing experience few symptoms even when they sit in a test room for 11 hours with the ozone levels up to 0.30 ppm—considered to be in the "very unhealthful range" in official smog alerts. On the other hand, a person who is exercising vigorously could suffer some breathing problems at ozone levels far below 0.30 ppm. But even here, the acute health effects are fairly minor.

For example, one study found that healthy people intermittently exercising vigorously for over two hours in air with 0.12 ppm of ozone suffered, on average, less than a 5% loss in lung function. This loss of lung function means that a person would experience only a mild to moderate cough,

Mr. Merline, a contributing editor to CR, *is a Washington correspondent for* Investor's Business Daily.

and would completely recover in less than 30 minutes, according to an extensive 1989 report from the Office of Technology Assessment (OTA).

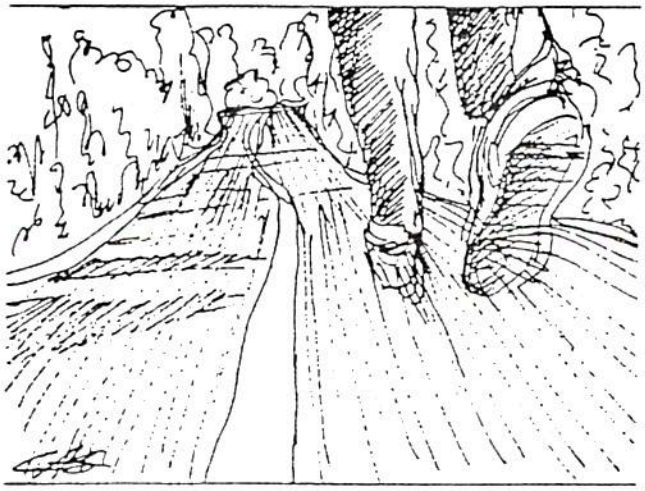
Other tests had subjects exercise for longer periods of time. In one, subjects worked out for six hours in a room with only 0.08 ppm of ozone. They suffered an 8% loss of lung function. But that's still in the mild range according to the OTA report, which reviewed the then-available medical literature on the subject.

Another study had testers exercise for more than six hours in a room with 0.12 ppm—the federal standard. They suffered an average 12% loss of lung function, a moderate effect, according to the OTA, characterized by a mild cough, shortness of breath, and some pain on deep breathing. The effects completely wear off in less than six hours. Still another study had subjects exercise for four hours in 0.16 ppm ozone. They experienced a 17% loss of lung function—still in the moderate range, according to the OTA.

Even among people exposed to high levels of ozone, most recovered quickly. Moderately exercising adults in a room with 0.35 ppm ozone suffered severe breathing problems—a 21% loss of lung function—characterized by repeated cough, moderate to severe pain on deep breathing, and some breathing distress. But lung function had basically returned to normal after 18 hours.

Other, more recent studies have looked at the picture from a different angle. Rather than conduct controlled experiments in test chambers with pure ozone, they have studied ozone levels in the real world and hospital admissions for respiratory problems. The goal of these studies is to determine whether high ozone levels are related to emergency room visits.

One—by researchers at the Nelson Institute of Environmental Medicine in New York and the EPA—found a relationship between "summer haze air pollution" and an "increased incidence of respiratory hospital admissions" in Toronto. Another found that emergency visits by asthmatics in central New Jersey were 28% higher during high ozone days than low ozone days. In this study, a high-ozone day was set at above 0.06 ppm (only half the federal limit), suggesting that asthmatics may be adversely affected by ozone levels below the current federal standard. Still, because these studies could not strictly isolate ozone, some of the effects may have been caused by other pollutants in the air. And the magnitude of the effects was small; in New Jersey, for example, "an additional 1.07" emergency visits on average occurred on days when the ozone was higher than 0.06 ppm.



Of those living in areas with smog problems, only half are ever exposed to ozone above the standard. And of this half who are exposed, only one-tenth are doing something that would cause any effect from ozone.

A headline-grabbing study published this past spring claimed to have found a statistically significant correlation between air pollution and cardiopulmonary and lung cancer-related deaths. "Sulfate and fine-particulate air pollution were associated with a difference of approximately 15% to 17% between mortality risks in the most polluted cities and those in the least polluted cities," noted this study, by researchers at Harvard University and the American Cancer Society. But this study did not attempt to isolate the effects of ozone. Instead, it focused specifically on the so-called fine particulate pollution—such as soot, smoke, and sulfate particles—an unrelated type of pollution largely produced by electric utilities and industry, but also by automobile exhaust. In any case, its results should be considered cautiously; in general, scientists warn that when a risk factor is as low as 17% it becomes extraordinarily difficult to say for certain that something else isn't causing the increased death rates—what scientists call confounding variables. That is, polluted cities might have some other factor, unaccounted for, that could cause the additional deaths.

Chronic Effects. While short-term ozone effects are generally mild, what happens if people are exposed to high levels of ozone over a long period of time, such as those living in the Los Angeles basin or near Houston, Tex.—areas with chronic smog problems? Health experts fear that long-term exposure to ozone could permanently damage the tissue of the lung, in effect quickening the

lung's aging process. Repeated exposures to high ozone levels could compound this problem.

So far, however, studies on this are inconclusive. Some suggest that inflammation of parts of the lung tissue could hasten the aging process, but nobody is certain. The Nelson Institute's Morton Lippmann, in a 1993 review of the literature on the health effects of ozone, observes that "several population-based studies of lung function indicate that there may be an accelerated aging of the lung associated with living in communities with persistently elevated ambient ozone, but the limited ability to accurately assign exposure classifications of the various populations in these studies makes a cautious assessment of these provocative data prudent."

Interestingly, scientists discovered that the body develops a defense mechanism against ozone. According to Lippmann: "Successive days of exposure of adult humans in chambers of ozone lead to an adaptation of lung function." After five days, there is a negligible effect from the ozone on lung function. He adds that "chronic seasonal human exposures to ambient air appear to produce a functional adaptation which persists for at least a few months after the end of the ozone season, but which dissipates by the spring."

A recently published study adds further weight to this finding. The study, by the independent Health Effects Institute in Cambridge, Mass., exposed laboratory rats to high levels of ozone for 20 months to see what damage this would do to the rodents' lungs. The results: "Ozone exposure had little or no measurable impact on lung function." In fact, the study found that the rats' lungs appeared to adapt to the ozone, protecting them from damage. What this means for humans is unclear. However, as the authors note: "Evidence from previous animal and human studies supports this suggestion that prolonged ozone exposure may lead to some degree of tolerance."

Another study attempted to determine whether ozone might cause lung cancer. This one, by the National Institute of Environmental Health Sciences, a federal agency, found no evidence of cancer in rats exposed for two years at ozone levels more than eight times as high as the federal standard.

Who Is At Risk?

How ozone is measured also may tend to exaggerate the health threat. While the federal government says that more than 100 million people are living in areas that violate the federal ozone standard, far fewer may actually be exposed to unhealthy air. Consider how an area violates the standard.

To measure ozone, the EPA uses monitors set up around the country. Heavily populated regions often have many monitors scattered throughout an area. An area violates the EPA standard if *one* of its monitors records ozone levels above 0.12 ppm on more than one day in a year. But it's not even an entire day. All that is required is for one monitors to tip the scales for an hour on a given day.

The EPA takes an average of three years to determine whether an area violates the standard. So an area could have two violations in one year, and one in each of the next two years, and violate the standard.

In other words, Detroit could be in violation of the EPA's ozone standard if the ozone level at its worst monitor peaked above the limit for a total of four hours spread over three years! And that's just at the worst monitor. Other parts of Detroit might not have been as bad. Of course, many areas are blanketed with high ozone levels for long periods over many days. Still, using the EPA's method can distort the magnitude of the prob-

People Exposed to Unhealthful Smog Levels*

(Based on 1983–85 air-monitoring data; Current estimates would be lower, but show similar proportions of exposure)

Exercise level	People exposed per year	% living in areas exceeding 0.12 ppm	Hours of exposure per person exposed per year
Nationwide except Los Angeles:			
Low	24 million	20%	3.7 hrs.
Moderate	16 "	13%	4.6 hrs.
Heavy	10 "	8%	3.2 hrs.
Very heavy	60 thousand	less than 0.1%	2.1 hrs.
Los Angeles:			
Low	9.7 million	97%	22 hrs.
Moderate	4.6 "	46%	24 hrs.
Heavy	3.0 "	30%	14 hrs.
Very heavy	20 thousand	0.2%	10 hrs.

* Estimates are based on hourly ozone data for the period 1983–85 and take into account people's activity patterns (e.g., time commuting, time indoors, etc.) and location throughout the day. The estimates are broken down according to exercise levels. Those exercising at the higher levels are most apt to be susceptible to health affects. The total number of people residing in areas where ozone concentrations exceeded 0.12 ppm at least one hour per year, on average during this time period, was approximately 130 million people. The Environmental Protection Agency, as of the end of 1994, estimates about 100 million people reside in ozone nonattainment areas. Although the population is smaller, the proportions should be similar for illustrative purposes.

SOURCE: Office of Technology Assessment, June 1989.

The Cost of Cleanup

The relatively minor health effects from ozone are in stark contrast to the enormous expense currently under way to cut ozone pollution. The 1990 Clean Air Act imposed costly new mandates on areas deemed to be in violation of the federal ozone standard. Among them were mandates for new centralized emissions testing facilities. Other areas may try to force the sale of electric vehicles, which emit no pollutants while driving (see article, page 10). Some of these mandates, most notably the centralized testing requirement, are being challenged by state governments. Still, the potential costs could climb as high as $13 billion a year.

To get a sense of what all these mandates would buy, experts at Resources for the Future, an environmental research group, tried to calculate the value of reduced ozone pollution. They determined that people would be willing to pay only about $1 billion a year for the health benefits derived from cutting ozone pollution. "The costs of proposed new controls are found to exceed the benefits, perhaps by a considerable margin," was their study's understated conclusion.

The costs could be even more out of line with the benefits. As an Office of Technology Assessment report notes, many people stay inside on high ozone days, not because of the ozone, but because of the intense heat that often accompanies it. So many people won't realize any health gain from a cut in ozone pollution, simply because they wouldn't be exposed in any case.

Worse, many of the controls the Environmental Protection Agency (EPA) has drawn up for cities with ozone problems may not work as advertised. Critics of centralized emissions tests, for example, argue that they have failed to cut pollution by anywhere near the levels the EPA says they will. The idea behind the tests is to catch high-polluting cars and force their owners to get needed repairs. To get even more pollution out of the air, the EPA wants many communities to switch to an enhanced version of the test, called I/M 240, which is both more costly and more time-consuming for car owners.

But the theory hasn't worked in the real world, according to some studies. Congress held hearings earlier this year at which several experts testified that the centralized testing program was a failure. Researchers at the University of Minnesota, for example, presented findings from their study of air pollution in Minneapolis/St. Paul before and after a centralized program was implemented. They found no discernible decrease in air pollution after the program started. (For a detailed discussion of the failure of centralized testing, see "Auto Emissions Tests Don't Work," *CR*, May 1994.)

lem—a fact the EPA acknowledges when asked.

Of those living in nonattainment areas for ozone, only half are ever exposed to ozone above the standard, according to Tom McCurdy, an analyst with the EPA. These people either live in areas within a "nonattainment" region where ozone never gets above the standard, such as Santa Monica, Calif., or they simply are indoors. (Ozone concentrations are significantly lower inside buildings than outside.) Of this half who are exposed, McCurdy adds, only one-tenth are doing something which would cause any effect from ozone. In other words, only about 5% of the people in regions experiencing elevated smog levels may be at risk of health effects. Those who tend to be in this group—and therefore the groups to be concerned about—are outdoor workers and children exercising outdoors.

The table at left, based on 1989 estimates compiled by the OTA, indicates the relatively small amount of time people may be exposed to such elevated ozone levels in a given year. (The most serious areas then, and now, were isolated within the Los Angeles region.) As the OTA notes, "ozone in a city's air...does not necessarily equal ozone in people's lungs. Concentrations vary with time of day and exact location. People vary in the amount of time they spend indoors."

Finally, it should also be noted that ozone pollution is dropping steadily—and will continue to fall—even without some of the costly new mandates. The reason is primarily that newer, less polluting cars continually replace older ones. As this fleet turnover continues, the air will get increasingly cleaner, absent any new air pollution mandates. Some experts note that fleet turnover alone will cut ozone pollution by up to 25% over the next 10 years. That would mean almost all areas outside California would be in compliance with the current federal ozone standard. And given the nature of smog, only a handful of days per year in the remaining areas will present a small group of people with a potential health threat. CR